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ON THE DIRECTION AND MANIFEST SIZE OF THE VARIATIONS OF POTENTIAL IN THE HUMAN HEART AND ON THE INFLUENCE OF THE POSITION OF THE HEART ON THE FORM OF THE ELECTROCARDIOGRAM

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GENERAL CONSIDERATIONS

(1) THE position of the heart influences the form of the ECG. Nevertheless, it is our main purpose in electrocardiographic investigation to study better the action of the heart, and one understands easily that if, by a change of position of this organ, an alteration in the form of the curve has been produced, difficulty will be encountered in deciding about the activity of the heart by means of this form.

(2) This difficulty can best be resolved if one previously has learned to recognize exactly the influence of position.

(3) The simplest and at the same time the most striking example of the influence of change of position on the form of the ECG is furnished by a case of situs inversus viscerum. Waller has already studied such cases, and several others have since been published. We are reproducing (Fig. 1) a curve taken with Lead I from a 7-year-old boy with situs inversus viscerum.

(4) One sees immediately that the peaks of Fig. 1, compared with those of a normal ECG, are reversed. If one changes the right- and the left-hand leads of the boy, there develops a curve which cannot be distinguished from a normal ECG and which is reproduced in Fig. 2. Figs. 1 and 2 are complete mirror images of each other.

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RESPIRATORY MOVEMENTS

(5) While the ECG in situs inversus viscerum can be explained in a very simple manner and requires no further discussion, in other cases of change in position of the heart phenomena appear which are more complicated and which must be analyzed more completely to be understood. We shall first consider those changes in position of the heart which are associated with respiratory movements.

(6) Most respiratory movements in woman as well as in man are to a lesser or greater extent diaphragmatic, and, as the diaphragm rhythmically assumes a high and a low position, the heart must also rhythmically be displaced within the thorax.

Fig. 1.

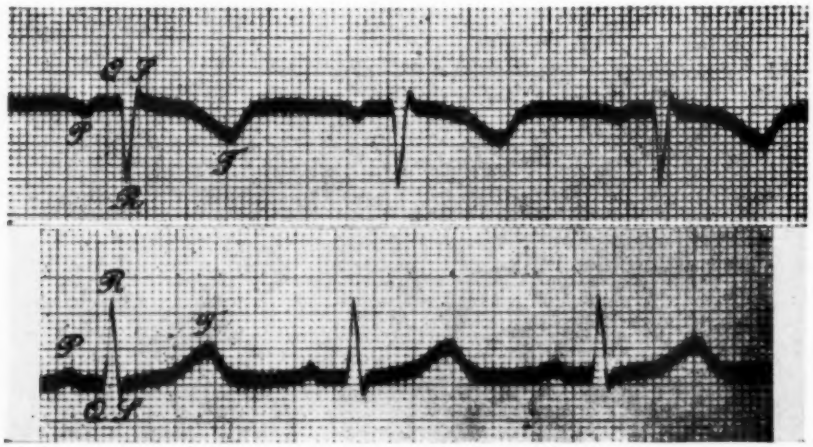


Fig. 2.

Fig. 1.—A case of situs inversus viscerum in Lead I. Abscissae, 1 division = 0.02 sec.; ordinates, 1 division = 10^{-4} volts.
Fig. 2.—As Fig. 1, with this difference only, that the right and left hands were exchanged in connecting with the galvanometer.

(7) Even before the construction of the string galvanometer, Samojloff[1] had already demonstrated in his lectures, with the aid of the capillary electrometer, that exaggerated respiration has a definite influence on the amount of the displacement of mercury. In Lead I the excursions become greater during the phase of expiration and smaller in the phase of inspiration.

(8) Among the numerous other investigators who have studied the influence of respiratory movements on the form of the ECG we can mention especially Vaandrager,[2] Kahn,[3] and Grau.[4]

(9) In many individuals the influence of the usual normal respiration is hardly noticeable, and at times one sees that even definitely deepened respiratory movements remain without remarkable consequences. One sees an example of this in a patient who has been described before and who showed the Cheyne-Stokes phenomenon[5] in a high degree. Periods of superficial, hardly noticeable respiratory movements alternated regularly with periods in which the patient was strongly dyspneic and made deep, vigorous, frequent respiratory movements.

ECG's taken from this patient during periods of apnea and dyspnea were hardly distinguishable from each other.

(10) In contrast to these results, there occur in other people even during normal respiration regular changes in the ECG which have the same rhythm as respiratory movements and therefore must be most intimately associated with them. If respiratory movements are deep enough, striking changes appear in all individuals.

(11) To study this more closely we carried out a systematic investigation on ten healthy men whose ages varied from 19 to 65 years. As an index of the depth of respiration, the vital capacity of each individual was measured previously, and he was instructed to make several deep inspirations and expirations in a Hutchinson spirometer during the photographic registration of the ECG. The amount of air to be displaced was to be about two-thirds of the vital capacity. This amount of respiratory movement was chosen because on the one hand it was large enough for our purposes, and on the other hand it presented no difficulties to the experimental subject.

Fig. 3.

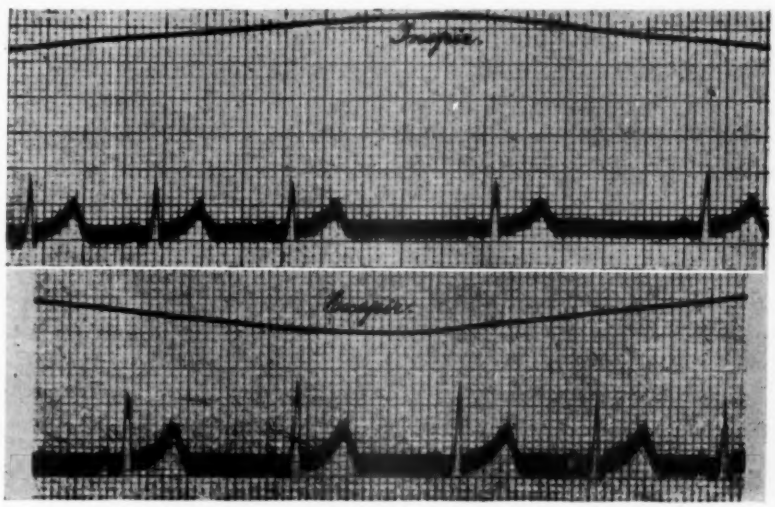


Fig. 4.

Fig. 3.—Bl. Lead I. During inspiration. Abscissae, 1 division = 0.04 sec.; ordinates, 1 division = 10^{-4} volts. The upper curve is the pneumogram.
Fig. 4.—Another portion of the same photographic record as the previous figure, during expiration.

(12) In Figs. 3 and 4 one can see two portions of a record taken with Lead I from Bl., one of the ten subjects.[6] Movements of the spirometer into which he breathed were registered on the photographic plate by means of a special arrangement of pulleys in such a manner that an upward movement of the writing arm corresponded to an inspiration and a downward movement corresponded to an expiration. The writing arm moved exactly perpendicularly in front of the slit of the registering device, and one scale division corresponded to 250 c.c. of inspired or expired air.

(13) The pneumogram, that is, the upper line in the two figures, shows waves whose amplitude varies between 11.3 and 11.5 scale divisions; the movement of air between maximal inspiration and maximal expiration thus amounts to 2.8 to 2.9 L., while a figure of 4.4 L. was obtained in a preliminary measurement of vital capacity in the subject.

(14) The pneumogram reproduces the changes in volume of the thorax without a significant delay, as can be proved by the tremors which the subject shows at the height of inspiration. One sees the tremors clearly appearing in the ECG. This is caused by the vigorous muscular exertion which takes place in extreme inspiration. As soon as inspiration is ended, the muscles relax and passive expiration begins; the tremors in the ECG stop. We noticed that this arrest of tremor corresponds almost exactly to the peak of the curve of the pneumogram in Fig. 3, whereby for our purposes we can easily neglect small differences of, for instance, five scale divisions (0.2 second).

(15) Apart from the form and the height of the peaks of the ECG, it is striking that the rate of the heart is subject to great differences during respiration. In Figs. 3 and 4 this is clearly in evidence, and at the same time one sees that the maxima and the minima of heart frequency do not coincide with the peaks of the pneumogram. Thus, for instance, the longest heart pause is found at the end of Fig. 3 and the shortest pause at the beginning of the same figure, although in both places the filling of the lungs with air is about the same amount. Thus also in Fig. 4 the filling of the lungs is the same at the beginning and at the end of the figure, while the frequency of the heart in these positions shows significant differences.

(16) The explanation of the phenomenon is known: during slow, deep respiration the gas content of the blood varies in sufficient amount to influence the tonus of the vagus nerves, in particular at the point where they take origin in the medulla oblongata, and since the maximum and the minimum of the vagal tonus do not correspond with the extremes of inspiration and expiration, the times of the least and the greatest heart frequencies must, in general, be displaced in relation to these positions.

(17) The displacement is not equally great in all men. In some of our experimental subjects the maximum of heart rate is present at the height of inspiration, in others at the depth of expiration, while in still others it shows up at various intermediate phases of respiration.

(18) We have mentioned this circumstance here particularly because it must be taken into account if the influence of respiratory movements on the form of the ECG is to be discussed. For we know that by means of the vagus nerves the form and the height of the T wave and especially of the auricular P wave can be altered significantly.

(19) In Figs. 5 and 6 we reproduce two portions of a tracing which was taken with Lead III from the same person. Here the influence of vagus stimulation on the P wave is clearly portrayed. After long heart pauses P is small, sometimes diphasic, sometimes entirely negative, while after the short intervals, that is, with greater frequencies and diminished vagal tone, P regains its normal shape and size.

(20) The maximal changes in P are not synchronous with the changes in the form of the ventricular EG. In Fig. 5, for instance, the first and the third heart beats show ventricular waves which can hardly be told apart, while P is positive in the first heart beat and negative in the third.

(21) Further, it is apparent from Figs. 5 and 6 that the maximal changes in P are not synchronous with the extremes of inspiration and expiration. This is perhaps demonstrated best in another curve (see Fig. 7). The center of the figure corresponds roughly with the position of extreme inspiration, as is apparent from the respiratory tracing. To the right and left of the center the inflation of the lungs with air is roughly the same, while the heart rates are very different. On the left side where the cardiac interval is short, one sees that a high P wave is present; on the right side of the figure where the heart pause is long, a low P wave occurs.

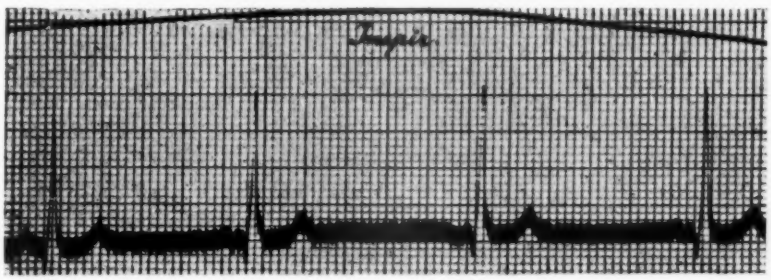


Fig. 5.—Bl. Lead III. During inspiration. The upper curve is the pneumogram.

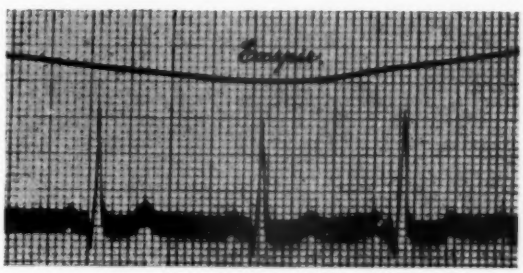


Fig. 6.—Another portion of the same photographic record as the previous figure, during expiration.

(22) It follows from the above considerations that one runs into difficulty if one wishes to discover by means of respiratory movements the influence to which the form of the ECG as a whole is subjected by a change of position of the heart, for the P wave is exposed to many influences during respiratory movements.

(23) In the discussion which follows, we shall attempt to separate these influences from each other and make it possible to discuss each separately, but for the present it is better to turn our attention to some other waves of the tracing.

(24) It is evident from Figs. 3 to 6 that in Lead I the peaks of the ventricular EG. become greater with expiration and smaller with inspiration, while in Lead III, on the contrary, they become smaller on expiration and larger on inspiration.

If we consider this fact in relation to the formula, Lead III = Lead II − Lead I, we must conclude that in Lead II the alterations in the waves of the ECG are insignificant. This is the case in Bl. as well as in most of the individuals studied, which leads to the practical conclusion that one should employ this lead in a patient when one wishes to study the form of his ECG as free as possible from the influence of respiratory movements.

(25) It must, nevertheless, be emphasized that even in Lead II noticeable variations in the size of the waves at times come to light, opposite in direction to the changes in Lead I. A typical example of such behavior is shown by Wi., whose ECG in Lead II is reproduced in Figs. 8 and 9. One sees how the respiratory tracing, whose waves show an amplitude of approximately 10.5 scale divisions, runs always very near the peaks of the R waves. A line drawn through the actual peaks of the R waves would run almost parallel to the pneumogram. The figures show that the waves increase during inspiration and diminish during expiration. The quantity of air displaced with each respiration amounts to 2.6 L., while the vital capacity of the experimental subject concerned amounts to 4.1 L.

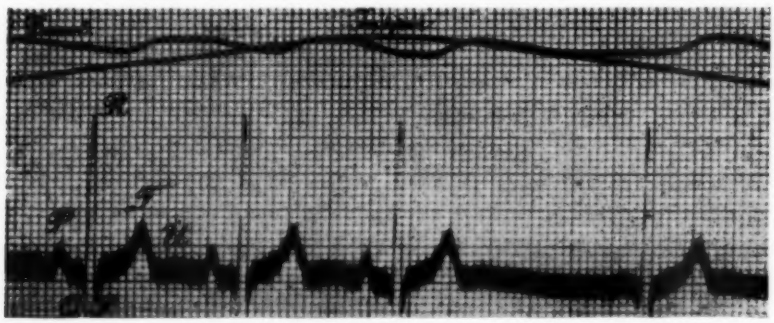


Fig. 7.—Br. Lead II. The respiratory tracing reaches its highest point at *Inspir.* *Carot.*, carotid pulse.

(26) In all of the foregoing figures from 3 to 9, the R and T waves are approximately equally diminished or increased during the various phases of respiration, so that the height relationships of the waves in a given person and in a given lead remain unchanged, and the tracing thus retains in general its characteristic appearance.

(27) For instance, when we measure out the waves of Figs. 3 and 4, we find in the position of maximum inspiration R = 6, T = 2.8 and $\frac{R}{T} = 2.14$; in extreme expiration R = 11, T = 0.5 millivolt, and $\frac{R}{T} = 2.20$. The relation of $\frac{R}{T}$ is thus nearly the same in both circumstances.

(28) But similar changes were not observed in all individuals. In some, one finds exceptions which on superficial observation appear to be completely irregular and thereby become somewhat puzzling.

(29) We will first describe some of these exceptions separately and then attempt to unite them in a single point of view.

(30) First, we point out that in many cases the change undergone by R can be significantly greater than the alteration in T. As examples we reproduce Figs. 10 and 11. Fig. 10,*A* and *B* shows the ECG of Bak. taken from Lead I. Two portions were cut out of the original tracing, and *A* represents a recording in inspiration and *B* in expiration. One sees that in expiration the R wave is significantly more increased than T, whereby the tracing takes on a completely different shape.

(31) This inequality becomes even more marked in Fig. 11,*A* and *B*, taken from Wi. Both parts are again reproductions of portions of one and the same tracing which was taken in Lead I; *A* corresponds to inspiration and *B* to expiration of the subject. In the first position R is smaller than T; in the second the relationship is reversed.

Fig. 8.

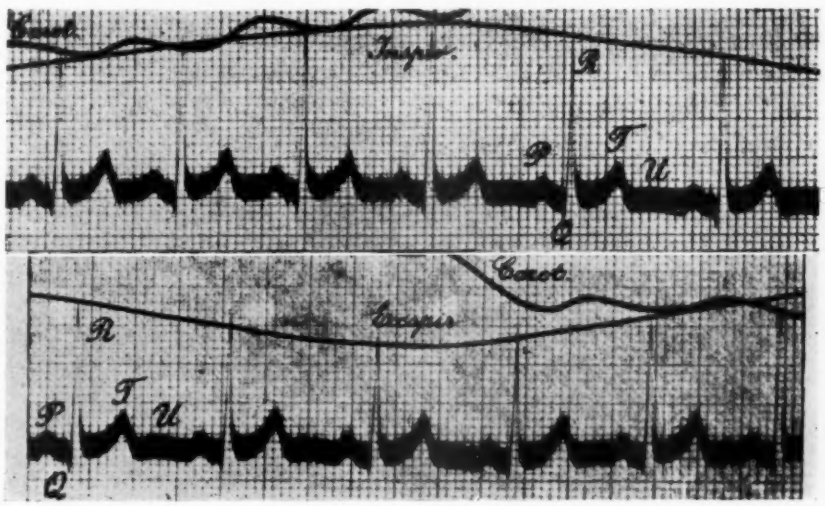


Fig. 9.

Fig. 8.—Wi. Lead II. *Carot.*, carotid pulse. During inspiration.

Fig. 9.—Another portion of the same photographic record as the previous figure, during expiration.

(32) Another exception consists in this, that in the same phase of respiration a wave that grows in Lead I also becomes larger in Lead III. This phenomenon appears when the waves in the two above-mentioned leads point in opposite directions. As examples we point to Fig. 12,*A* and *B*, which reproduces the ECG in Lead III of the same person, Bak., from whom also the tracing of Fig. 10 is taken. The T_{III} wave is here negative, and we see that during expiration T_{III} (Fig. 12), as well as T_I (Fig. 10), become larger.

(33) Entirely similar considerations are applicable to the QRS group as to T.

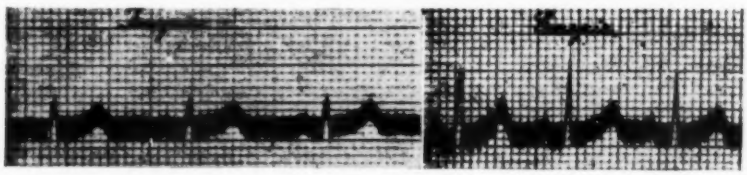
(34) We will, nevertheless, attempt to answer the question of how the apparently irregular influence of respiratory movements on the form of the ventricular EG. can be explained.

(35) We notice, first, that without exception in the persons studied the maxima and minima of the waves coincide exactly with the extreme respiratory positions, and that therefore one ought to take this phenomenon into consideration in every attempt at an explanation. Second, we exclude the possibility that such potential differences as appear in our curves are generated by some epiphenomenon, as, for example, muscle action related to the respiratory movement or movements of the electrodes. For in case some such accidental cause exerted an influence synchronous with respiration, the tracing reproducing a series of successive ECG's would move rhythmically up and down as a whole. We see, however, that the line that could be drawn through the diastoles of the heart shows no such rhythm but, in fact, as can be easily established with the help of the square millimeter grid, runs completely straight in many photographs.

(38)

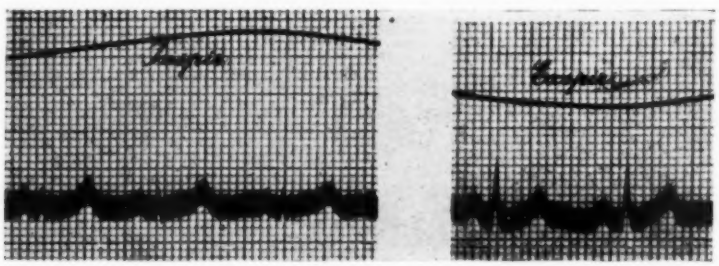
(39)

(40)



A. B.

Fig. 10.—Bak. Lead I. Fig. 10, *A* and *B* are portions of one and the same continuous photographic record. Fig. 10, *A* during inspiration; Fig. 10, *B* during expiration.



A. B.

Fig. 11.—Wi. Lead I. Fig. 11, *A* and *B* are portions of one and the same continuous record. Fig. 11, *A* during inspiration; Fig. 11, *B* during expiration.

(41)

(36) As a second possibility to be considered, we mention the influence of altering body resistance to which Samojloff[7] has called attention. We assume that during respiration the resistance of the body between the leads remains constant. If very small variations did occur here, they could be neglected without hesitation in view of the great resistance of the galvanometer. But during respiration the electric resistance of the organs surrounding the heart must undergo rhythmic variations on account of the altered air and blood content of the thorax, and these must necessarily exert an influence on the amplitude of the waves of the ECG. Apparently this influence is by itself very small, as is indicated, in our opinion, by the fact that the tracing in Lead II undergoes in general only small changes while the changes seen in Lead III are usually opposite those obtained in Lead I.

(42)

(43)

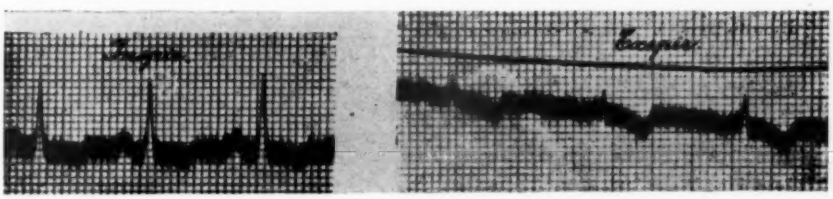
(37) Were change in resistance the cause of the alterations in amplitude of the waves, one would have to assume that in a respiratory movement the resistance

of the thorax diminished in the horizontal direction while at the same time it increased in the vertical direction and remained unaltered in the oblique direction. Such an assumption seems to us very unlikely, especially because in not too vigorous respiratory movements the expansion of the thorax in the various directions is pretty much the same.

(38) Also, the fact that the relation of the size of the waves often undergoes a significant change is not explainable on the basis of a change in body resistance. Samojloff has already pointed this out himself, and he rightly concludes that in any event change in resistance cannot be the sole cause of the phenomenon.

(39) Although on physical grounds we must assume a priori that an influence of changing body resistance does exist, the above analyses justify the conclusion that it is small and for practical purposes may be disregarded in most cases.

(40) As a third cause, we can point out that the manner and way in which the heart contracts can be influenced by the altered pressure relations within the heart as well as in the thorax. The negative intrapleural pressure, which is increased during inspiration and diminished during expiration, exerts a definite direct mechanical influence upon the circulation. On account of its weaker musculature the right heart will be more sensitive to this influence than the left, and if the activity of one-half of the heart is more or less changed than that of the other, then the QRS group in the ECG would in particular be modified. It is, in fact, this group that undergoes the clearest changes during respiration.



A. B.

Fig. 12.—Bak. Lead III. Fig. 12, *A* and *B* are portions of one and the same continuous tracing. Fig. 12, *A* during inspiration; Fig. 12, *B* during expiration.

(41) For an exhaustive study of the influence which extreme changes of intrathoracic pressure can exert on the ECG, we direct attention to the investigations of Kahn,[8] who made ECG's of men while they carried out the Valsalva experiment. The changes thereby appearing in the ECG were described by Kahn as very significant.

(42) Nevertheless, in ordinary and even in quite deep respiration, pressure relationships do not constitute the main reason for the changes in form and amplitude of the ECG, for they cannot explain the above-described, and apparently irregular, variations.

(43) We discuss finally the influence of changes in position which the heart undergoes in the thorax during respiration. This influence has already been brought to the foreground by Grau,[9] who carried out his investigations especially on "men with movable hearts," in whom he controlled the position of the heart roentgenologically.

(44) Even earlier we had considered it not at all surprising[10] that the rhythmic changes in position of the heart caused by respiration should also modify rhythmically the form of the ECG. Apparently the change in position caused by normal respiration is alone insufficient in most people to evoke noticeable changes in the form of the ECG. This fact, which is reinforced by the circumstance that occasionally quite deep respiration remains without notable effect, is by itself something remarkable. For the respiratory motions are but seldom purely thoracic, and the anatomic relations make it necessary for the heart to experience a change in position in abdominal respiration. Transilluminating the human chest with roentgen rays, one sees these changes in position come clearly to view. (52)

(45) Still more questions emerge when one wishes to explain the changes in form of the ECG by alterations in position of the heart. How is it possible that the several waves often become greater or smaller quite unequally? Why does the ECG show so much more inconsiderable modifications in Lead II than in Leads I and III?

(46) All these difficulties are resolved at a single stroke if one does not restrict oneself to the form of the registered tracings, but attempts from the ECG as recorded by the various leads to derive the direction and manifest size[11] which the resulting potential differences in the heart itself possess.

RELATIONSHIP OF THE TRACINGS TAKEN BY THE THREE CONVENTIONAL LEADS

(47) Before we go on to describe the manner and means whereby one can ascertain the resultant potential difference within the heart, it is desirable to review in a few words the mutual relation which exists among the three leads now generally employed.

(48) The formula,[12] Lead III = Lead II − Lead I, has validity only if the current flowing through the galvanometer exerts no influence on the potentials present at the extremities. Now this factor is so small that it can be neglected without causing any noticeable error. For the total resistance of the circuit in which are found the galvanometer and the human body is very large in comparison with the resistance of the heart musculature. We can thus assume a priori that the formula mentioned is correct.

(49) On superficial inspection of many curves some doubt of the practical applicability of the formula seems to arise. This concerns particularly those ECG's which show great variations in form in the three leads. One can compare, for instance, the tracings of Fl. published previously (Figs. 13, 14, and 15).

(50) These and similar curves give a false impression with respect to their mutual relationship, which is caused by the circumstance that often the waves that we are accustomed to designate with the same letter do not fall in the same phase of a cardiac cycle. One can demonstrate this in various ways, among others by comparing the distances between the peaks P, R, and T in the tracing from Lead I with the corresponding distances in the tracings from Lead II and Lead III. It is apparent then that the summits mentioned are in general closer to each other in Lead I. (53) (54)

(51) That our formula is in complete agreement with fact can be shown without too great difficulty. One would gain one's objective most simply if one recorded

two or three tracings from the several leads simultaneously on the same photographic plate. A simultaneous double registration has already been carried out by other investigators, who, as far as I know, employed two small-model string galvanometers and did not attempt an exact measurement of the tracings. An oscillograph is not suitable for the purpose mentioned.

(52) Another method has been employed by Fahr.[13] He recorded, as did Kahn, the ECG and the heart sounds simultaneously. In one and the same person the ECG was recorded from the three leads under identical conditions, while the tracing of heart sounds was combined with each ECG. In this way a fixed point of time could be marked, which fell always in the same phase of a cardiac cycle. With the aid of this fixed point of time the phases of the tracings in the various leads could then be mutually identified with exactness.

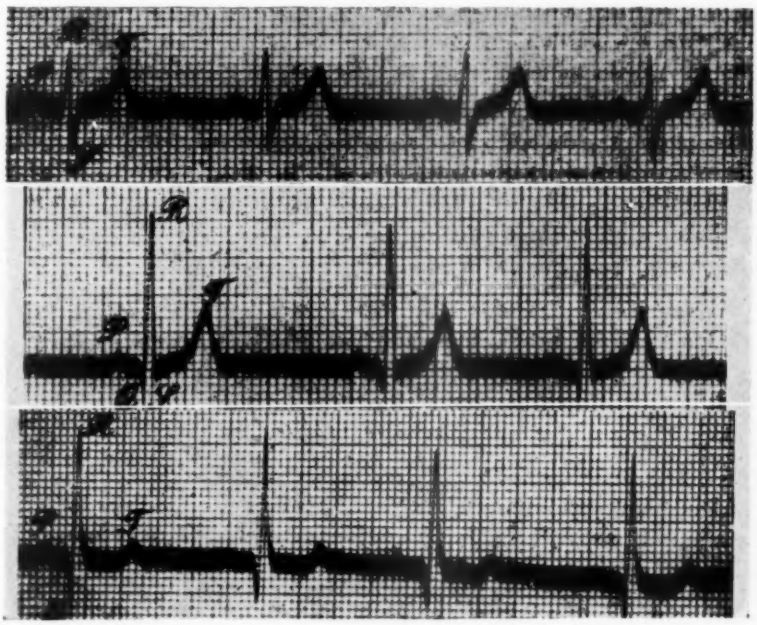


Fig. 13.

Fig. 14.

Fig. 15.

Fig. 13.—Fl. Lead I.
Fig. 14.—Fl. Lead II.
Fig. 15.—Fl. Lead III.

(53) The investigation of Fahr shows that the ventricular EG. as a rule begins earlier in Leads II and III than in Lead I, and that the summit of R_I is formed somewhat earlier than the summits of R_{II} and R_{III}. Making use of this data it is no longer difficult to demonstrate the validity of the formula even in apparently complicated forms of the ECG.

(54) As an example we reproduce here in Fig. 16, the QRS group of the ECG of Fl. (cf. Figs. 13, 14, and 15), schematically and in one and the same coordinate system. In its construction the abscissae are, for the sake of clarity, four times more extended than the ordinates, so that the whole figure seems expanded in breadth: one scale division on the abscissa corresponds to 0.01 sec.; one scale

division on the ordinate corresponds to 10^{-4} volts. The vertical lines represent various phases of a cardiac cycle, and at each phase the rule holds: Lead II − Lead I = Lead III.

(55) One sees that the waves Q_{II} and Q_{III} begin 0.01 sec. before R_I while the summits of the first-named waves correspond with a point of the anacrotic portion of the latter wave. The entire wave S_I corresponds to the catacrotic part of R_{III}, while the whole wave S_{II} corresponds with the anacrotic part of S_I.

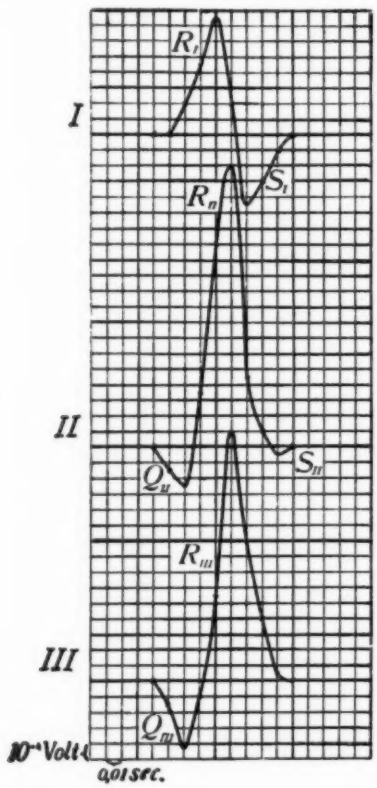


Fig. 16.—Construction of the QRS group of Fl. in the three leads. Each ordinate corresponds to identical phases of a cardiac cycle. Abscissae, 1 division = 0.01 sec.; ordinates, 1 division = 10^{-4} volts.

(56) Similar constructions which are capable of clarifying our understanding of the significance of the QRS group can always be carried out without great difficulty and also applied to the P and T waves. One must then, however, always bear in mind that these constructions lose their value if one changes the conditions under which the three leads are taken. Understandably one must give the subject the same body position during the recordings, while the scale divisions of the three systems of coordinates must correspond to the same time intervals and the same potential differences. Further, the galvanometer must react quickly enough to indicate the potential variations to be registered, immediately and without a significant error.

(57) In conclusion, still another remark should be made regarding the calculation of such tracings. Inasmuch as absolute precision can naturally never be achieved and small errors, say of the order of a few per cent, can always occur, the accuracy with which one derives a small low wave by subtraction of two waves of nearly the same size will be very small.

(58) If, for instance, T_I and T_{II} are nearly of equal size, the calculated value of T_{III} from the formula, $T_{III} = T_{II} - T_I$, will be very inexact. T_{III} as actually recorded then gives its size more exactly than the calculated, and in a comparison of the three tracings it is proper in these circumstances to start from one of the first two and the third lead in order to derive the tracing of the remaining lead from the data obtained hereby.

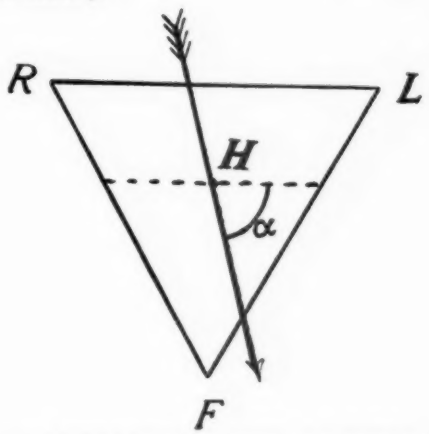


Fig. 17.—Schema of the equilateral triangle. *R* corresponds to the right hand, *L* to the left hand, and *F* to both feet. The heart *H* is located in the center. The arrow indicates the direction of the potential difference in the heart. $\alpha = 76°$.

(59) The above discussion makes it clear that one should not as a rule content oneself with two leads in the belief that the tracing of the remaining lead is sufficiently known from the data of the two former. For the dimensions of a curve constructed by calculation can be derived not only more simply but also more exactly by direct registration.

THE SCHEMA OF THE EQUILATERAL TRIANGLE: DIRECTION AND MANIFEST SIZE OF THE RESULTING POTENTIAL DIFFERENCE IN THE HEART

(60) How, from the tracings taken in the three leads, is one to determine the actual direction of the potential difference in the body?

(61) If one attempts to answer this question, one attains the goal in the simplest way by schematizing the human body. The following schema, that can be well-designated as the schema of the equilateral triangle,[14] can be recommended as especially useful. Herein the human body is represented as a flat, homogeneous plate in the form of an equilateral triangle, *RLF*, (Fig. 17). Current is led off to the galvanometer from the corners. *R* corresponds to the right arm, *L* to the left arm, while *F* represents the potential of both feet. A lead from *R* and *L* corresponds, therefore, to Lead I, from *R* and *F* to Lead II, and from *L* and *F* to Lead III.

(62) A small spot, *H*, in the middle of the triangle, represents the heart. We assume that at a certain instant the potential differences in the heart are so distributed that their resultant has the direction of the arrow portrayed in the figure. A lead from the heart in this direction shows a maximal potential difference of such a nature that in the direction of the head of the arrow the heart is positive, in the opposite direction, negative. (73) (74)

(63) We can also represent the matters thus: that between two closely adjacent points of the small spot *H* a potential difference is developed. The arrow drawn in the figure coincides with the line which joins these two points and represents the direction of the maximal potential difference in the heart. The distance separating the points is very small compared with the length of one side of the triangle. (75)

(64) The angle that the arrow makes with the side *RL* is called α and reckoned as positive when the arrow is turned in the clockwise direction and negative when turned in the opposite direction. In determining this the standpoint of an observer who faces the anterior chest wall of the subject is assumed.

(65) We now assume further that an ECG has been recorded from a subject that has in each of the three leads a simple form, so that the summits R_I, R_{II}, and R_{III} fall at identical phases of a cardiac cycle. According to the well-known formula, it can then be easily verified that $R_{II} - R_I = R_{III}$.

(66) If we transfer the values found in the experimental subject over to the schema, we can determine in him the direction of the potential difference which was present in the heart during the registration of the R wave, and thus was the cause of the formation of this wave.

(67) The ECG of Bak. in inspiration can serve as an example. One finds in the three leads the following values. These are expressed in tenths of a millivolt: $R_I = 3.2$, $R_{II} = 12.5$, $R_{III} = 9.3$.

(68) In the homogeneous equilateral plate which contains the heart at its center, these values can only be produced by a resulting potential difference whose direction coincides with the arrow drawn in such a manner that $\alpha = 76$ degrees.

(69) In expiration there is found in the same person $R_I = 9.2$, $R_{II} = 11.2$, $R_{III} = 2.0$, from which one can calculate that under these circumstances $\alpha_1 = 40$ degrees (cf. Fig. 18). (76)

(70) By means of the schema we are in this manner in a position to show that during expiration the heart has rotated itself in the body around a sagittal axis. The extent of the rotation in the schema is $\alpha_1 - \alpha = -36$ degrees. (77)

(71) In the construction of our schema we have assumed that the heart lies as a material point in a homogeneous mass and that the distances of the heart from the three leads and also the resistances concerned are equally great. This is, understandably, not the case. The electrical resistance to conduction in the lungs is different from that in the heart itself and in the chest wall. Only one point is assumed in the schema for both feet, although small but still measurable potential differences are produced between left and right foot.

(72) Nevertheless, we have sufficient reason to assume that the actual rotation of the heart in the body for practical purposes corresponds sufficiently exactly with the rotation in the schema, and therefore in the following lines we shall (78)

identify the direction which the potential difference assumes in the human body with the direction of the arrow in the schema and designate it as α.

(73) The manner and way in which one calculates the angle α from the measured potential differences will be elucidated more closely in the appendix. We mention here only that the calculation is simple and can be carried out quickly.

(74) It is possible to attempt to verify the results thus found by means of an investigation with roentgen rays. Nevertheless, great demands on roentgen technique are made if one wishes to measure exactly the angle by which the axis of the heart turns during respiration. One would have to be able to make instantaneous exposures which correspond with a definite phase of the cardiac cycle, as, for example, they have been carried out by Koranyi and v. Elischer.[15]

(75) Further, it must be noted that in different men the motility of the heart shows great differences and that therefore for achievement of our goal the roentgenograms and the ECG's must be taken in the same person and under the same conditions. A few experiments which we have carried out in this direction have not progressed far enough to be discussed here in greater detail.

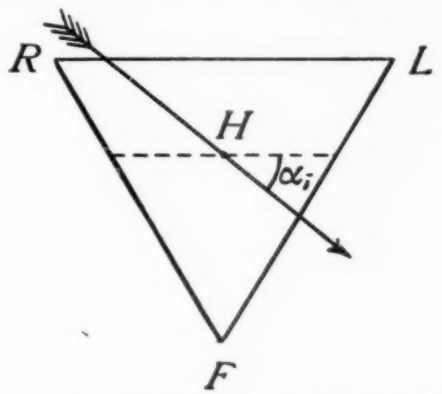


Fig. 18.—For explanation of the illustration, see Fig. 17. α_1 = 40 degrees.

(76) In the roentgenogram the direction of the heart's axis can be determined with any certainty only with difficulty, and in so far as we are now aware, nowhere in the whole of the roentgen literature is a detailed, decisive treatment of the question of the rotation of the axis of the heart encountered.

(77) Nevertheless, data have been published which are not without value for our purpose. We mention especially here the book of Groetdel,[16] in which a cinematogram of the heart in forced respiration is reproduced. Groedel comes to the conclusion that in respiration the left heart border is displaced more strongly than the right, namely in the proportion of 6.5 to 4.8. The heart thus rotates itself, so to speak, around a point which lies at the place where the right diaphragm and the right auricle meet.

(78) In general, these results agree well with the results of electrocardiography. They cannot, however, be a satisfactory control on this latter because the rotation of the cardiac axis can be measured with greater accuracy electrocardiographically than roentgenographically.

(79) The schema of the equilateral triangle not only gives the direction of the potential differences, but it places us also in the position to make a comparison between the magnitudes of these differences as they are found in the heart itself. For clarity we can introduce a designation "the manifest potential difference in the heart" and, when particular waves, for instance, P, R, or T, are considered, indicate them by the figures P_m, R_m, and T_m. By these figures, then, the manifest value of each summit is meant.

(80) We define the manifest potential difference in the heart as the magnitude given in one of the three leads when the direction of the current between the leads corresponds to the direction of the resulting potential difference in the heart. Only the potential differences in the frontal plane come into consideration therein.

(81) If we assume that a given potential difference in the heart produces the summits T_I, T_{II}, and T_{III}, and that the direction of current in the schema is represented by an arrow parallel to the side *RL* (Fig. 19), then T_I is the greatest of the three summits while T_{II} and T_{III} will be opposed to each other and attain half the size of T_I. In these circumstances $T_m = T_I$.

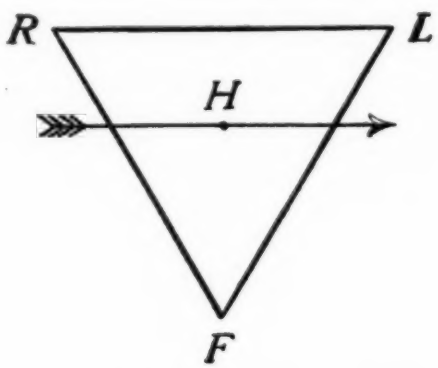


Fig. 19.—For explanation of the illustration, see Fig. 17. α = 0 degree.

(82) The manifest size of a wave can always be calculated from the height of the recorded wave and can thus always be expressed in absolute terms, for instance, in tenths of a millivolt.

(83) The calculation, just as that of the angle α, can be carried out easily and rapidly, as will be more completely described in the appendix. Applying the calculation to the R wave of Bak., the data of which have been given previously, (see Figs. 10 and 12), we find that $R_m = 13$ in inspiration and in expiration $R_m = 12$ tenths of a millivolt.

(84) In Table I the similar values of four subjects are collected.

(85) One sees from the figures recorded in the table that the rotation of the resulting potential difference R, present in the heart, takes place in the same direction in all subjects during respiration, but, however, as was to have been expected, the extent of this rotation is not the same in the various people. The rotations vary from $-12°$ to $-36°$.

TABLE I.

SUBJECT	R_I	R_{II}	R_{III}	DIRECTION OF R AND ROTATION IN EXPIRATION	MANIFEST SIZE OF R IN TENTHS OF A MILLIVOLT (R_m)
	TENTHS OF A MILLIVOLT				
Bak., inspiration	3.2	12.5	9.3	76° } −36°	13
Bak., expiration	9.2	11.2	2.0	40° }	12
Bat., inspiration	6.2	20.0	14.0	73° } −20°	20
Bat., expiration	12.2	20.2	8.0	53° }	20
de Bl., inspiration	6.0	26.0	20.0	77° } −12°	27
de Bl., expiration	11.0	26.0	15.0	65° }	26
Br., inspiration	6.5	21.0	14.5	73° } −23°	21
Br., expiration	13.0	20.0	7.0	50° }	20

(86) The manifest size of R which we have called R_m remains unchanged during respiration in one and the same person, within the limits of error of the method, although it shows great differences in various people. In Bak. it has a value of approximately 12.5, and in Bl., of about 26.5×10^{-4} volts.

(87) The constancy of the manifest size of R in the same person demonstrates, first, that the influence of altering body resistance on the size of the peaks of the ECG is but slight, and, second, it can be considered as a proof of the validity of the schema employed.

(88) We must now determine the direction and the manifest size of T in the same way as for R, but must remember that the T wave is not suitable to bring to light clearly the change of position of the heart caused by respiration. The changes which T shows during respiration have two causes, of which the first is associated with the change in position, the second with the change in frequency of the heart. It is not difficult, however, to determine from the data of the tracings the part which each of the two causes has in the modification of T.

(89) Attention should be first directed to the manifest size of T. It is apparent from Table II that T_m regularly increases during expiration, and, in our subjects, from a minimum proportion of 1.2 to a maximum proportion of 1.9.

(90) In accord with the schema of the equilateral triangle, increase or diminution of the manifest size of a wave can never be produced by a rotation of the axis of the heart. Neither will a displacement of the heart noticeably influence the manifest size of a wave when it is confined to normal limits. A change of this magnitude must therefore be ascribed exclusively to other influences, and as one such we have come to recognize the action of vagal tonus with which the heart rate is also associated.

(91) The rotation of a wave, however, is a different matter than the manifest size. We know from the data of Table I how much the axis of the heart will be rotated in the four subjects mentioned during the respiratory movements carried out by them and measured by the spirometer. The data of Table II concern the same respiratory movements as those of Table I and are taken from the same

TABLE II.

SUBJECT	T_I	T_{II}	T_{III}	DIRECTION OF T AND ROTATION IN EXPIRATION	MANIFEST SIZE OF T IN TENTHS OF A MILLIVOLT (T_m)	RELATION OF MANIFEST SIZES OF T ($\frac{T_m \text{ IN EXPIRATION}}{T_m \text{ IN INSPIRATION}}$)
	TENTHS OF A MILLIVOLT					
Bak., inspiration	2.0	1.5	−0.5	16°	2.1	1.9
Bak., expiration	4.0	1.5	−2.5	−8° −24°	4.0	
Bat., inspiration	1.5	2.5	1.0	53°	2.5	1.2
Bat., expiration	2.0	3.0	1.0	49° − 4°	3.1	
de Bl., inspiration	2.7	5.0	2.3	57°	5.0	1.4
de Bl., expiration	5.0	6.5	1.5	43° −14°	6.85	
Br. inspiration	3.0	5.0	2.0	53°	5.05	1.2
Br., expiration	4.5	5.5	1.0	40° −13°	5.9	

ECG. If one therefore finds a different angle for the rotation of T than for the rotation of the axis of the heart, the difference between the two rotations must be caused by the action of additional influences. In our experiments only the changeable vagal tone can be held responsible for the latter.[17]

(92) As a matter of fact, we find values for the rotation of T which are mostly significantly smaller than for the rotation of R. Thus, for instance, in Bak. a value for the rotation of R, that is, for the rotation of the heart axis, of $\beta_R = -36$ degrees is found, while in the same ECG a value for the rotation of T of $\beta_T = -24$ degrees is found. The difference $\gamma = \beta_T - \beta_R = 12$ degrees is then the rotation caused by the altered vagal tone.

(93) It is apparent that γ is, as a rule, positive, whether or not the manifest size of T increases. One can receive confirmation of this finding by taking the ECG of a number of people in whom the vagal tonus is changed in other ways than by forced breathing. For instance, one can study the subject at rest and immediately after strenuous physical exertion when the heart frequency is increased.

(94) The results of such an investigation, which will be reported in the material which follows, confirm the above-mentioned conclusions.

(95) P is as little suitable as T to indicate the changes in position of the heart caused by respiration, for P is even more greatly influenced by a change in heart rate than T. In addition, P is often small and less simple. In so far as it was possible to determine the direction of this auricular current in our ten subjects, it indicated without exception that during respiration a rotation takes place in the same sense as with R and T.

(96) The small and rather inconstant peaks Q and S occur mainly where the QRS group is complicated so that their measurement is rendered difficult. But the same thing can be said of them as of P. Where it was possible to carry out the measurement and to draw a not too uncertain conclusion from small modifications, the same result was always found, that the two waves rotate in respiration in the same direction as the other waves of the ECG.

(97) The cause of all these rotations taking place in the same sense is clear. For the whole heart is turned around a sagittal axis by the change in position of the diaphragm during respiration.

(98) It would not be without interest to ascertain whether the axis of the heart retains the same direction in diastole and systole. During diastole the heart has, to all intents and purposes, no tendency to assume a characteristic shape and is able to follow the movements of the diaphragm better than during systole. P falls in a phase where the ventricles still remain in diastole, while the QRS group is produced in the very beginning of ventricular contraction and apparently therefore falls in such a phase of this contraction where the musculature has not yet entirely attained its systolic hardness. Finally, T falls in a phase where the ventricles show their full hardness.

(99) Were one able to determine exactly the direction of each wave and the influence which respiration exerts on its rotation, it would be possible to determine separately the rotation of the heart's axis in diastole and in systole. But the difficulties of measurement of P and the complication because of altered cardiac frequency do not permit one to determine with certainty whether the rotation of P and the QRS group caused by change of position are as marked or more marked than those of T.

(100) It is better to leave this point undecided and to remark further that during respiration no noteworthy rotation of the heart takes place around a vertical axis. This is proved by the behavior of the peak S_I. As will soon be discussed more fully, S_I shows in some individuals very great modifications during rotation of the heart around the vertical axis, while this wave is only slightly changed during respiration in the same individual.

(101) Finally, we return to the question brought up in the beginning, and apparently presenting many difficulties, but which now can be answered easily.

(102) First, the question, how is it possible that in ordinary respiratory movements in which roentgenographic study often demonstrates clear changes in position of the heart, one usually observes no significant changes in the ECG?

(103) The explanation must be sought therein, that during an ordinary, not excessive respiration in many cases there occurs mainly a displacement of the heart parallel to itself. Such a displacement exerts only a slight influence on the form of the ECG while a small rotation of the heart on a sagittal axis as it is produced during a deeper respiration already suffices to modify noticeably the form and height of the various waves of the tracing. This arises directly out of the schema of the equilateral triangle.

(104) That the various waves of the ECG are often unequally increased or diminished by an altered position of the diaphragm, and that the tracing in Lead II shows so much less important modifications than in Lead I and III, is explained in the most easy and satisfactory manner by the triangle schema. A potential difference present in the heart, whose direction according to the schema would be designated in the one heart position by $\alpha = 80$ degrees and in the other by $\alpha_1 = 40$ degrees, produces a wave which in Lead II remains unchanged and in Leads I and III on the contrary would be modified significantly.

(105) If the wave in Lead II in the first heart position had an amplitude of 9.4×10^{-4} volts, it would retain this value in the same lead in the second heart position. In Lead I, on the contrary, it would be increased from 1.7×10^{-4} volts in the first heart position to 7.7×10^{-4} volts in the second position of the heart. In Lead III it would be diminished from 7.7×10^{-4} volts in the first heart position to 1.7×10^{-4} volts in the second heart position.

(106) When the resultant potential difference T has a different direction than R, the waves T_I, T_{II}, and T_{III} must be modified in a different manner than R_I, R_{II}, and R_{III} by the rotation of the heart around a sagittal axis. Therefore, in general, it is not permissible to conclude from the fact that T behaves differently under certain conditions than R, that the development of electricity whereby T is produced has another cause than the development of electricity whereby R is evoked.[18]

(107) It is likewise clear why a wave which is directed similarly in Leads I and III undergoes opposite changes through a change in position of the diaphragm, while another wave is increased in both leads or diminished in both leads, as long as in the former it has a positive and in the latter a negative value. The latter takes place when the angle α lies between -90 degrees and $+30$ degrees.

THE INFLUENCE OF CHANGE IN POSITION OF THE BODY

(108) There exist good grounds to assume that the most important peaks of the ventricular EG., T and R, are produced by potential changes which are localized in approximately frontal planes of the heart. This can be demonstrated by the behavior of the S wave in Lead I. When a person lies first horizontally on his left side and later turns to his right side, the striking phenomenon appears that the S_I wave, which in the first position was not present at all or was only very small, appears clearly and sometimes reaches a considerable height when the body is turned.

(109) Figs. 20 and 21, which were both taken from Lead I, are examples of the development of a large S wave upon turning the body to the right side. Fig. 20, which was recorded during the left-sided position, shows a small S wave which can almost be overlooked, while Fig. 21, which was taken from the right-sided position, shows so large an S wave that one is inclined to wonder somewhat that both tracings were taken from the same person with the same lead, and, with exception of body position, under completely similar circumstances.

(110) The most satisfactory solution of this phenomenon, so puzzling on superficial consideration, will be found if one assumes that on turning the body from the left to the right side the heart is rotated in the thorax around the longitudinal axis of the body.

(111) When there is present in the heart a potential difference having a sagittal direction, a small rotation of the heart around the long axis of the body can cause a definite change in the amplitude of the waves involved, and this will occur especially markedly in Lead I. In case the potential difference is directed exactly sagittally, the wave disappears completely. It is clearly apparent that the great differences in the S wave in various individuals as well as its general inconstancy must be explained by this circumstance.

(112) In Figs. 20 and 21 we notice that the P, R, and T waves undergo almost no change. If we conclude from the enormous modification of the S wave that a rotation of the heart has taken place along the longitudinal axis of the body, we must also assume that the P, R, and T waves are produced in approximately the frontal plane, or better, in such planes as form relatively large angles with the sagittal plane. For in this manner it is easiest to explain their slight variability under these circumstances.

(113) The immutability of the P, R, and T waves gives occasion for another remark. Anatomic relationships require that in turning the body to the left side, the heart is displaced in the thorax somewhat to the left; on turning the body to the right side, it is displaced somewhat to the right. That this change of position evokes no modification in the P, R, and T waves confirms the result which investigation of respiration yielded, namely, that a displacement of the heart parallel to itself exerts only a very minor influence on the form of the ECG.

Fig. 20.

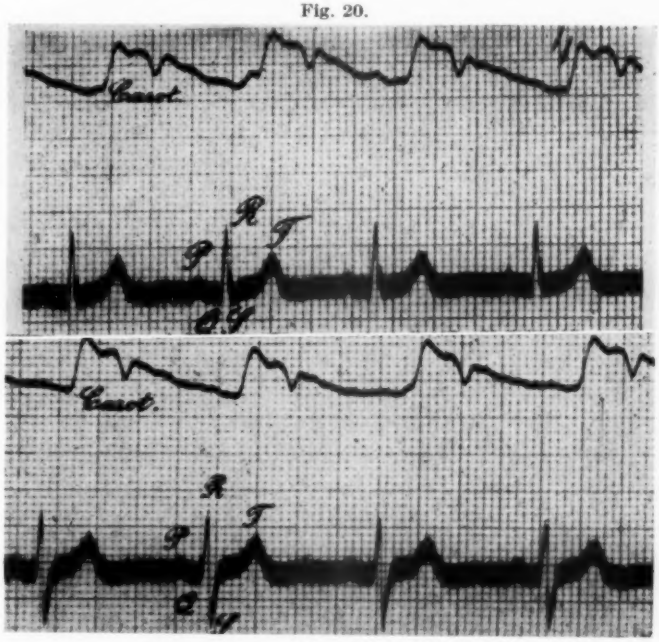


Fig. 21.

Fig. 20.—Fl. Lead I, patient lying on the left side.
Fig. 21.—The same person with the same lead, lying on the right side.

(114) In five of the ten persons studied, as well as in the above-mentioned case of Fl., the S_I wave became greater as soon as the body was turned from the left to the right side. In three subjects S_I was lacking in both positions of the body, and in the two others this wave retained the same size.

(115) When we compare the ECG's which were taken from men lying upon the back and upon the abdomen successively, it appears that in turning over on the abdomen the heart rotates on a sagittal axis in the same sense as in expiration. The ten individuals studied showed this rotation without exception.

TABLE III.

SUBJECT	FREQUENCY OF HEART IN BEATS PER MINUTE	TENTHS OF A MILLIVOLT			DIRECTION OF T AND ROTATION WITH INCREASED HEART RATE	MANIFEST SIZE OF T IN TENTHS OF A MILLIVOLT (T_m)	RELATION BETWEEN MANIFEST SIZES $\left(\frac{T_m \text{ AT INCREASED HEART RATE}}{T_m \text{ AT REST}}\right)$
		T_I	T_{II}	T_{III}			
Ca. at rest	72	5.0	4.5	−0.5	25°	5.5	
Ca. after exercise	112	5.0	7.0	2.0	46° 21°	7.2	1.3
v.d.Sch. at rest	76	4.0	5.0	1.0	41°	5.3	
v.d.Sch. after exercise	97	6.0	8.0	2.0	44° 3°	8.3	1.6
Fu. at rest	86	1.5	2.0	0.5	44°	2.1	
Fu. after exercise	137	2.5	3.5	1.0	46° 2°	3.6	1.7
Tj. at rest	91	3.0	3.3	0.3	35°	3.6	
Tj. after exercise	131	3.5	5.5	2.0	51° 16°	5.6	1.6
Jo. at rest	89	3.0	4.2	1.2	46°	4.3	
Jo. after exercise	96	3.0	7.0	4.0	65° 19°	7.0	1.6

(116) The explanation imposes no difficulties, for in the prone position pressure in the abdominal cavity is increased and through the pressure of the abdominal viscera against the diaphragm this organ is pushed upward.

(117) Apart from the rotation around the sagittal axis there can also be recognized in the same circumstances, by the behavior of S_I, a minor rotation around the longitudinal axis of the body.

(118) We mention now briefly the differences which the ECG shows when an individual is studied in the sitting position and while lying on the back. In the ten persons studied the differences were in general but slight; in some it was clear that the heart in the recumbent position had rotated on a sagittal axis in a similar manner as in inspiration and in the sitting position in a similar manner as in expiration. In others these rotations were, however, hardly noticeable.

(119) Because this question has a practical importance for the clinic, it must be recalled here that in our investigation the trunk of the subject was really horizontal during recumbency and vertical while seated. As a rule, one is not able to fulfill these conditions in the clinic. For when a patient is studied seated, one permits him to lean back comfortably in the chair—at least we ourselves always proceed in this way, generally also with a patient lying in bed, the shoulders are at a somewhat higher level than the pelvis.

(120) In this way the two body positions approach each other so that the difference between the ECG's becomes smaller, and, as a rule, one can without much hesitation compare directly the tracings registered while lying with those recorded while sitting.

THE INFLUENCE OF INCREASED HEART RATE

(121) With the help of the schema of the equilateral triangle one is in a position better to form an opinion about the form of the ECG in all sorts of circumstances. For instance, let us consider more closely the influence of an increased heart rate caused by bodily exertion. In "Weiteres über das ECG" a number of this kind of tracings have already been described,[19] but the description at that time had to be restricted to the separate ECG's taken from the three leads. Now we can combine the results, and, for instance, deduce from the measurements of P_I, P_{II}, and P_{III}, that the manifest size of the potential difference present in the heart, P_m, increases with increasing heart rate, therefore with diminished vagal tonus.

(122) Worthy of notice is the change that the ventricular EG. undergoes under the influence of diminished vagal tonus. We point especially to the changes in the T wave and give in the following Table III the height reached by this wave in five persons.[20]

(123) From the values in the sixth column it is evident that in all of the cases studied the direction of T is rotated a bit in the positive direction when the heart rate increases, while the seventh and eighth columns indicate that then the manifest size of T also increases.

(124) The investigation is fairly troublesome. For the construction of the above table it was necessary to measure out six ECG's for each subject, or thirty ECG's in all. So long as one studies the person in the resting state his heart rate is as a

rule relatively constant during the recording of the three leads. But if one takes the photograph immediately after bodily exertion while the heart rate is increased, a difficulty arises because it is not always possible to catch the right moment when the frequencies are the same. It should not remain unmentioned here that the ECG's of v.d. Sch. and Fu. appear from the table to satisfy these requirements better than those of the three other subjects who have therefore not furnished as reliable results.

(125) The rotation of T, which, as is apparent from the table, is positive in all persons studied, is not caused by a rotation of the heart's axis. In the first place, there is no reason for the assumption that under the above-mentioned conditions a rotation of the axis of the heart took place, and second, the unaltered direction of this axis can be proved directly by measurement of the R wave.

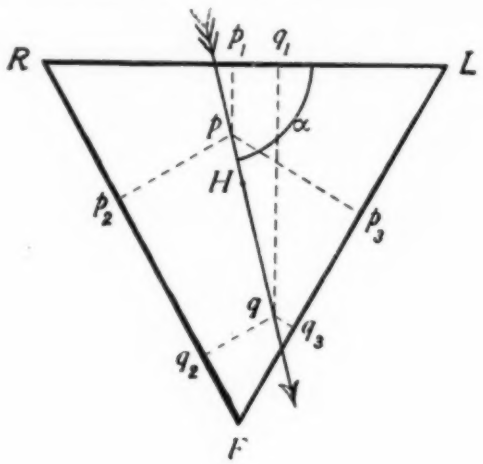


Fig. 22.

(126) The positive rotation as well as the increase in manifest size of T therefore depends exclusively upon the increase in the heart rate and must be explained by the diminution of the vagal tonus. Vagal tonus thus influences the path which excitation and the wave of contraction take in the heart muscle.

PATHOLOGICAL CONDITIONS

(127) In conclusion, several examples from pathology can be discussed.

(128) In myodegeneratio cordis and in many cases of auricular fibrillation T is small in all three leads. It need not be discussed in greater detail that under these circumstances T_m must also be small.

(129) In many cases of hypertrophy of the left heart one finds a negative QRS_{III}. What conclusion can one draw from this? Entirely in general, the modification of the QRS group could perhaps be caused by a change in position of the heart, especially by a rotation of the heart around the sagittal axis. In fact, the clinician is often in doubt whether he should ascribe the phenomena seen in his patients to an actual hypertrophy or simply to a change in the position of the heart.

(130) The schema of the equilateral triangle in such a case can often provide the solution.

(131) We found, for instance, in several cases of hypertrophy of the left ventricle that the most important peaks of the QRS group, or at times the QRS group as a whole, had a direction which could be represented by $\alpha = -40$ degrees, while for this wave or group in normal hearts the amplitude of α varied between +40 degrees and +90 degrees.

(132) If one wished to explain the rotation of the QRS group in these cases by a rotation of the heart around its sagittal axis, one would have to assume that this rotation amounted to 80 degrees and even 130 degrees—which must be considered as impossible.

(133) The application of the schema permits therefore the conclusion that we are concerned in these cases with a significant change in the conduction of excitation and the progress of the wave of contraction through the heart muscle. It is entirely possible that one would be able to diagnose a beginning hypertrophy of the left heart through electrocardiographic investigation of such a change sooner than in any other existing manner.

(134) Hypertrophy of the right heart and also atypical contractions of the heart can be adjudged in the same manner. The latter are of great importance and worth a special exhaustive review. But the above-mentioned examples may suffice. They prove adequately that by means of the schema one is in a position to separate from each other the real changes in the heart's action and the apparent ones, which are caused only by change in the position of the heart.

APPENDIX

On the Manner and Way in Which One Calculates the Direction and Manifest Size of the Resulting Potential Difference

(135) *A. The Direction of the Resulting Potential Difference.*—In the equilateral triangle *RLF* in Fig. 22 an arrow is drawn through the center *H* making an arbitrary angle α with the side *RL*. On this arrow a portion *pq* of arbitrary length is marked off. The projection of *pq* on the side *RL* is p_1q_1, that on the side *RF* is p_2q_2, and that on the side *LF* is p_3q_3.

(136) If we make $pq = E$, $p_1q_1 = e_1$, $p_2q_2 = e_2$, and $p_3q_3 = e_3$, thus:

$$e_1 = E \cos \alpha \quad \ldots\ldots (1)$$
$$e_2 = E \cos (\alpha - 60 \text{ degrees}) \quad \ldots\ldots (2)$$
$$e_3 = E \cos (120 \text{ degrees} - \alpha) \quad \ldots\ldots (3)$$
$$e_3 = e_2 - e_1 \quad \ldots\ldots (4)$$

(137) We assume that the equilateral triangle *RLF* represents a homogeneous flat plate, that in two points lying very close to each other in the immediate neighborhood of *H* a potential difference is created, and that a line joining the two points together coincides with the arrow drawn in the figure. Under these circumstances the potential differences present between the corners of the triangle must be related to each other as $e_1 : e_2 : e_3$.

(138) When α is unknown, this angle can be calculated from the relationship of two of these three potential differences. Employing e_1 and e_2 in the calculation, one finds with the help of the formulas (1) and (2):

$$\operatorname{tg} \alpha = \frac{2e_2 - e_1}{e_1 \sqrt{3}} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (5).$$

If one employs e_1 and e_3, one finds:

$$\operatorname{tg} \alpha = \frac{2e_3 + e_1}{e_1 \sqrt{3}} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (6),$$

and employing e_2 and e_3, one finds:

$$\operatorname{tg} \alpha = \frac{e_2 + e_3}{(e_2 - e_3) \sqrt{3}} \quad \ldots\ldots\ldots\ldots\ldots \quad (7).$$

(139) The angle α represents the direction of the resulting potential difference in the body. By means of any one of the formulas (5), (6), and (7), one is also in a position to calculate this direction from the potential differences as these are found in the three conventional leads.

(140) Nevertheless, it is not always worth the recommendation to employ the above-mentioned formulas because one can often achieve his purpose much more quickly. If one is content with a lesser accuracy so that an error of 10 degrees or 15 degrees is still permissible, one can use a geometric construction whereby α is derived directly from the figure. One sees easily that with angles of 0 degrees, 30 degrees, 60 degrees, 90 degrees, etc., the relationship of $e_1 : e_2 : e_3$ is very simple and, in fact,

at 0 degrees	$e_1 : e_2 : e_3 = 1 : 0.5 : -0.5$
at 30 degrees	$e_1 : e_2 : e_3 = 1 : 1 : 0$
at 60 degrees	$e_1 : e_2 : e_3 = 0.5 : 1 : 0.5$
at 90 degrees	$e_1 : e_2 : e_3 = 0 : 1 : 1$ etc.

(141) The angles of values lying in between must then be estimated.

(142) If greater accuracy is desired, one can employ a table of figures to advantage. Such a table is Table IV. With its help one is in a position to read off the value of α to within a minute in any case that may occur. The added table of interpolation, Table V, gives directly the desired value for every two degrees.

(143) *Example 1*: For the R wave of Bak. in inspiration there were found in tenths of a millivolt: $R_I = 3.2$, $R_{II} = 12.5$, $R_{III} = 9.3$. We have thus for this wave: $e_1 : e_2 : e_3 = 3.2 : 12.5 : 9.3$. Since the table shows the relationship of potential differences for a maximum value of ± 10, we must multiply the three above figures by $\frac{10}{12.5}$ and obtain thus: $e_1 : e_2 : e_3 = 2.6 : 10 : 7.4$. It can be seen from the table that this proportion corresponds to an angle α which lies between 70 degrees and 80 degrees. As shown in the table the value of e_1 diminishes from 3.5 to 1.8 between 70 degrees and 80 degrees. An interpolation table which lies between those figures, 3.5 and 1.8, must therefore be consulted. Such a one is Table V, II.

TABLE IV. TABLE OF RECORDED AND MANIFEST POTENTIAL DIFFERENCES FOR VARIOUS VALUES OF α*

	RECORDED POTENTIAL DIFFERENCES (e)			MANIFEST POTENTIAL DIFFERENCES (E)
	e_1	e_2	e_3	
0°	10	5.0	−5.0	10.0
10°	10	6.5	−3.5	10.2
20°	10	8.2	−1.8	10.7
30°	10	10	0	11.5
40°	8.2	10	1.8	10.7
50°	6.5	10	3.5	10.2
60°	5.0	10	5.0	10.0
70°	3.5	10	6.5	10.2
80°	1.8	10	8.2	10.7
90°	0	10	10	11.5
100°	−1.8	8.2	10	10.7
110°	−3.5	6.5	10	10.2
120°	−5.0	5.0	10	10.0
130°	−6.5	3.5	10	10.2
140°	−8.2	1.8	10	10.7
150°	−10	0	10	11.5
160°	−10	−1.8	8.2	10.7
170°	−10	−3.5	6.5	10.2
±180°	−10	−5.0	5.0	10.0
−170°	−10	−6.5	3.5	10.2
−160°	−10	−8.2	1.8	10.7
−150°	−10	−10	0	11.5
−140°	−8.2	−10	−1.8	10.7
−130°	−6.5	−10	−3.5	10.2
−120°	−5.0	−10	−5.0	10.0
−110°	−3.5	−10	−6.5	10.2
−100°	−1.8	−10	−8.2	10.7
−90°	0	−10	−10	11.5
−80°	1.8	−8.2	−10	10.7
−70°	3.5	−6.5	−10	10.2
−60°	5.0	−5.0	− 0	10.0
−50°	6.5	−3.5	−10	10.2
−40°	8.2	−1.8	−10	10.7
−30°	10	0	−10	11.5
−20°	10	1.8	−8.2	10.7
−10°	10	3.5	−6.5	10.2
−0°	10	5.0	−5.0	10.0

*For each group of three leads the maximal value for the recorded potential differences is set at ±10.

(144) Here one finds for e = 2.5 (that is, for the value which lies closest to the value 2.6) the corresponding angle $\alpha = 6$ degrees. It follows therefrom that the value sought is $\alpha = 70$ degrees + 6 degrees = 76 degrees.

(145) *Example 2*: The following were found for the T wave of Bak. in expiration, in tenths of a millivolt: $T_I = 4$, $T_{II} = 1.5$, $T_{III} = -2.5$. We have thus for these waves: $e_1 : e_2 : e_3 = 4 : 1.5 : -2.5$. Since the table expresses the relationship of

TABLE V. INTERPOLATION TABLE

DIFFERENCES IN DEGREES		POTENTIAL DIFFERENCES (±e)	DIFFERENCES IN DEGREES	E
0°		0	10°	11.5
I	2°	0.4	8°	11.3
	4°	0.8	6°	11.1
	6°	1.	4°	11.0
	8°	1.5	2°	10.8
10°		1.8	0°	10.7
II	2°	2.2	8°	10.5
	4°	2.5	6°	10.4
	6°	2.9	4°	10.3
	8°	3.2	2°	10.2
10°		3.5	0°	10.2
III	2°	3.8	8°	10.1
	4°	4.1	6°	10.1
	6°	4 4	4°	10.0
	8°	4.7	2°	10.0
10°		5.0	0°	10.0

the potential differences in terms of a maximal value of ±10, the above figures must be multiplied by $\frac{10}{4}$. We get then: $e_1 : e_2 : e_3 = 10 : 3.75 : -6.25$. It can be seen from the table that this proportion corresponds to an angle α which lies between −10 degrees and 0 degrees.

(146) According to the information in the table, the value of e_2 decreases between 0 degrees and −10 degrees from 5 to 3.5. An interpolation table is therefore sought which lies between these values 5 and 3.5. Such a one is Table V, III.

(147) Here one finds for e = 3.8 (that is, a figure that lies closest to value 3.75) the corresponding angle equals 8 degrees. It follows from this that the value sought is $\alpha = -8$ degrees.

(148) It occurs but rarely that one has at his disposal data with which he can calculate α more exactly than to within 1 degree or 2 degrees. We have already pointed out that it is at times not easy to decide whether the potential differences which occur in any one of the three leads fall in exactly the same phase of a cardiac cycle. Further, there are unavoidable errors in measurement, which, when an adequate string galvanometer is used, are caused less by the imperfections of the instrument or by variations in body resistance than by difficulties in setting the sensitivity of the string exactly at a previously determined magnitude.

(149) We must, however, keep in mind the fact that errors are attached to the measurements, even if of only a few per cent. It is therefore often desirable to correct the results of direct measurement before one goes on to calculate the angle α. One takes care thereby that the corrected value always satisfies the

condition of formula (4), namely, that $e_3 = e_2 - e_1$, for only under these circumstances can it be calculated with the desired accuracy.

(150) The application of the correction can be illustrated by an example. If we assume that one finds for a given wave of the ECG, for example, for the T wave in the three leads, the following values expressed in tenths of a millivolt: $T_I = 10$, $T_{II} = 10$, $T_{III} = -1$. These values must be corrected. Assuming that the percentage of error in the amplitude of the waves in each of the three leads is about the same size, the absolute value of the error will be the least in the small wave T_{III}. After percentile correction and rounding out of hundredths of a millivolt, the amplitude of $T_{III} = -1.0$ remains unchanged, while T_I and T_{II} are modified in the sense that $T_I = 10.5$ and $T_{II} = 9.5$. With the help of these corrected values which now fit the requirement of formula (4), the angle α is calculated.

(151) It is worthy of notice that those ECG's are particularly suitable for the calculation of the direction of the resulting potential differences which have a simple form, and in which, for every wave, the formula (4) is almost completely applicable without correction. In ECG's of complicated form in which one can with difficulty recognize the identical phase of a cardiac cycle in the three leads, the calculation of α can be time consuming.

(152) Fortunately many simple forms occur in which, as has been pointed out above, the measurement can be profitably employed without a great expenditure of time.

(153) *B. The Manifest Size of the Resulting Potential Difference.*—The manifest size of the resulting potential difference is represented in Fig. 22 by the length of *pq*, which we have made equal to E. This value must be distinguished sharply from the actual potential difference in *H* itself. The relation between E and the potential difference in *H* itself is dependent upon the mutual distance apart of the two points lying in *H* between which the potential difference is developed.

(154) In our discussion we assume that the distance between the two points mentioned is small compared with the size of the triangle, because otherwise we have no way of accounting for this distance. For this reason nothing can be said of the size of the potential difference in *H* other than that compared with E it is very large.

(155) The value of E is to be calculated from the values which the leads from the corners of the triangle give. We express it in the angle α and in one of the three projections e_1, e_2, or e_3 and write thereby the formulas (1), (2), and (3) in the form

$$E = \frac{e_1}{\cos \alpha} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (7)$$

$$E = \frac{e_2}{\cos (\alpha - 60 \text{ degrees})} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (8)$$

$$E = \frac{e_3}{\cos (120 \text{ degrees} - \alpha)} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (9).$$

TABLE VI.

TIME IN SECONDS	POTENTIAL DIFFERENCES IN TENTHS OF A MILLIVOLT IN LEADS			RESULTANT POTENTIAL DIFFERENCE	
	I	II	III	DIRECTION	MANIFEST SIZE (10^{-4} VOLTS)
0.01	0	0	0	—	0
0 02	0	−1.5	−1.5	−90°	1.7
0.03	2.0	−2.5	−4.5	−64°	4.5
0.04	4.5	3.5	−1.0	18°	4.7
0.05	7.5	13.0	5.5	55°	13.0
0.06	2.0	18.0	16.0	84.5°	19.8
0.07	−4.5	4.5	9.0	120°	9.0
0.08	−3.0	1.0	4.0	136°	4.2
0.09	−1.0	−0.5	0.5	±180°	1.0
0.10	0	0	0	—	0

(156) For instance, when α has the value 0 degrees or ±180 degrees, the potential difference in *H* is parallel to the side *RL*. In these circumstances $pq = p_1q_1$ or $E = e_1$, and the manifest size of the potential difference is given directly by the amplitude registered in Lead I. In the case $\alpha = 60$ degrees, $E = e_2$.

(157) In the case $\alpha = 120$ degrees we get $E = e_3$.

(158) If one wishes to derive the value of E for any arbitrary value of α, one can use any one of the formulas (7), (8), or (9). By means of relatively simple formulas one can express the value of E directly in e_1 and e_2, e_1 and e_3, or in e_2 and e_3. It is, however, simpler to employ again the table of figures.

(159) *Example 1*: It was stated in Example 1, Section A, that, for Bak. in the state of inspiration, the values recorded were $R_I = 3.2$, $R_{II} = 12.5$, and $R_{III} = 9.3$ tenths of a millivolt while the direction of the resulting potential difference for the R wave was determined by means of the interpolation Table V, II as $\alpha = 76$ degrees.

(160) The value of 10.4 for the manifest potential difference corresponding to this angle is read off directly from the interpolation table concerned. But since the table gives the values in the instance that the maximum of the registered potential difference equals 10, while in our case in fact the maximal value of R_{II} amounts to 12.5×10^{-4} volts, the manifest size of R must be calculated as $R_m = \frac{12.5 \times 10.4}{10} = 13.0 \times 10^{-4}$ volts.

(161) *Example 2*: It was stated in Example 2, Section A, that for Bak. the recorded values in expiration amounted to $T_I = 4$, $T_{II} = 1.5$, $T_{III} = -2.5$ tenths of a millivolt, while the direction of the resulting potential difference of the T wave was determined by means of the interpolation Table V, III as $\alpha = -8$ degrees.

(162) The value of 10.1 for the manifest potential difference corresponding to this angle is read off directly from the interpolation table concerned. Since, however, the table gives the value for the case in which the maximum of the recorded potential difference, $e = 10$, while in our case the maximum value of T_I

actually amounts to 4×10^{-4} volts, the manifest size of T must be calculated as

$$T_m = \frac{4 \times 10.1}{10} = 4.0 \times 10^{-4} \text{ volts.}$$

(163) If one does not wish to use the interpolation table, one may read off the size of the manifest potential difference directly from Table IV accurately enough.

(164) The sign of the manifest potential difference should always be positive. One needs to introduce no negative values and should consider that the description of the manifest potential difference is complete when one states when it occurs, its direction, and its amplitude.

(165) In Table VI the directions and the manifest sizes of the QRS group of Fl., as it was constructed in Fig. 16, are recorded for each one hundredth of a second.

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5. Compare Weiteres, etc., ref. above, p. 564.
6. In these and all subsequent figures one scale division on the abscissa corresponds exactly with 0.04 sec., and one scale division on the ordinate with 10^{-4} volts.
7. See ref. above, p. 183.
8. See ref. above, p. 307.
9. See ref. above.
10. Compare, Weiteres über das E.K.G., p. 563.
11. For the meaning of the expression "manifest size" consult text to follow.
12. Compare, Weiteres über das E.K.G., p. 558.
13. Fahr, G.: On Simultaneous Records of the Heart Sounds and the Electrocardiogram, Heart, vol. 4, no. 2, p. 147, 1912.
14. Consult also a report given on March 19, 1912 at the Chelsea Clinical Society in London. *Lancet*, March 30, 1912, p. 853. [Reprint of report follows this translation.]
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16. Groedel, F. M.: Die Röntgendiagnostik der Herz- und Gefässerkrankungen, p. 68-70, Hermann Meusser, Berlin, 1912.
17. In considering this, no account is taken of the possibility that perhaps the mobility of the heart is greater in the state of diastole than in systole. Consult on this point the text a few lines further.
18. Consult on this point "Über die Deutung des E.K.G," Pflüger's Arch., vol. 149, p. 65, 1912.
19. See ref. above, p. 565 et seq.
20. The data are taken from the tracings employed in "Weiteres über das E.K.G."

(See following page for Concordance in Paragraphs Between Pfluger's and Lancet Articles.)

CONCORDANCE IN PARAGRAPHS BETWEEN PFLÜGER'S AND *Lancet* ARTICLES*

Pflüger's	*Lancet*	*Pflüger's*	*Lancet*	*Pflüger's*	*Lancet*
1	42	35	59	70	66
2	43	36	60	71	—
3	43	37–39	—	72	66
4	43	40	61	73	66
5	44	41	—	74–78	—
6	45	42	61	79	67
7	46	43–45	—	80	68
8	46	46	62	81	68
9–11	—	47	23	82	69
12	46	48	23	83	69
13	47	49	25	84	—
14	48	50	24	85	70
15	49	51	26	86	—
16	50	52	27	87	—
17	50	53	28	88	71
18	50	54	28	89–104	—
19	51	55	28	105	76
20–21	—	56	29	106–107	—
22	52	57	29	108	72
23	52	58	29	109	72
24	53	59	30	110	73
25	—	60	—	111	73
26	54	61	62	112	74
27	—	62	62	113	75
28	55	63	—	114–120	—
29	55	64	62	121	—
30	56	65	63	122–126	78
31	57	66	63	127–130	—
32	58	67	64	131–133	79
33	58	68	64	134	80
34	59	69	65		

*The concordance is at times exact with no changes other than different paragraphing. At other times, however, a single sentence in the *Lancet* article is expanded into several paragraphs in Pflüger's article, and new illustrative material is added. The impression is clear that the *Lancet* article was written first and was reorganized, edited, and enlarged for publication in Pflüger's *Arch. f. d. ges. Physiol.*

CONCORDANCE IN FIGURES BETWEEN PFLÜGER'S AND *Lancet* ARTICLES

Pflüger's (Fig. No.)	*Lancet* (Fig. No.)	*Pflüger's* (Fig. No.)	*Lancet* (Fig. No.)
1	14	12a	24
2	15	12b	25
3	16	13	5
4	17	14	6 not quite identical
5	18	15	7
6	19	16	8
7	—	17	26
8	—	18	27
9	—	19	28
10a	20	20	29
10b	21	21	30
11a	22	22	—
11b	23		

THE DIFFERENT FORMS OF THE HUMAN ELECTROCARDIOGRAM AND THEIR SIGNIFICATION[1]*

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(1) The application of electrocardiography to clinical examination gives valuable data in two different ways. In the first place, it shows the time relations between the action of the ventricles and the auricles. The difference in time between the contraction of these two parts of the heart can be measured easily and accurately. Diseases such as Stokes-Adams, in which the auricles have a rhythm different from that of the ventricles, and which sometimes give difficulties when examined by the older methods, are always immediately recognized electrocardiographically. And further it may be mentioned that a new insight has been obtained into several other diseases, among which auricular fibrillation takes a prominent place.

(2) But this evening we shall pass over this first group of symptoms, and we shall limit our attention to the second one, which is concerned with the form of the EKG. If this form is perfect, it must be the exact expression of the changes in potential difference which take place between two parts of the human body. It is self evident that a perfect form can only be obtained by means of a perfect instrument, and it need not be mentioned that such an instrument does not exist. The capillary electrometer is not rapid enough when made sufficiently sensitive, and therefore not suitable for clinical purposes. The oscillograph suffers from the same fault, although in a lesser degree, and has, moreover, the great disadvantage that the movements of the recording mirror are oscillatory. On the contrary, the string galvanometer is in general sufficient for clinical purposes, and by means of this instrument we are able to approximate the exact form of the EKG. so closely that the remaining divergences from it become practically imperceptible.

(3) Not every model of the string galvanometer is equally suitable. Even if we have at our disposal a good instrument in all other respects, the correctness of the work done with it depends in a high degree upon the qualities of a small part of the apparatus, the string, and we have further to take into consideration the magnification and the sensitiveness used.

(4) How is the clinician to know if the curves which he has obtained with the instrument are reliable reproductions of the electrical variations under investigation? He can, it is true, correct the curves obtained by means of data determined from the properties of the instrument, and which are to be found by means of control curves. But such a correction requires a fairly detailed calculation and a reconstruction of the curve, for which reason he will be loth to apply it. And happily this is unnecessary in most cases. It is only necessary to record a control curve in such a way that an equal resistance from a rheostat is introduced into the galvanometer circuit in place of the body resistance, which usually has a value of from 1000 to 2000 ohms.

(5) The tension of the string must be so regulated that an excursion of the image of the string of 1 mm. is equal to 10^{-4} volts. At a given moment a constant potential difference is thrown into the circuit. If the string reaches its new position of equilibrium within about 0.01 second or less, the instrument is rapid and at the same time sensitive enough for recording EKG.'s with sufficient accuracy. The deviations from an ideal EKG. which a curve recorded under these conditions exhibits are so small that in the great majority of cases they may be neglected.

(6) To prove this we reproduce in Figs. 1 and 2 two EKG.'s which were taken from the same person by lead I. The sensitiveness of the galvanometer was accurately adjusted in both records, so that 1 scale division corresponded to 10^{-4} volts. All other conditions were also kept the same as nearly as possible, with the one exception that in recording Fig. 2 a magnification was used twice as large as in Fig. 1. Therefore the string was stretched twice as much in Fig. 2, and in accordance with this the duration of a deflection was reduced to half.

(7) It is possible to determine the duration of a deflection to a certain degree by means of Fig. 3, which was recorded immediately after Fig. 2 upon the same photographic plate. A resistance of 2300 ohms was introduced into the galvanometer circuit from a resistance box, this resistance being equal to that found for the body of the person investigated under the conditions of the

[1] A paper read before the Chelsea Clinical Society on March 19th, 1912.
*Lancet. 1912[1], 853-861.

experiment. The introduction of a potential difference of 3×10^{-3} volts causes a deflection of 3×10 scale divisions, and each deflection is completed in about 0.5 scale divisions—i.e., 0.01 second. The control curve for Fig. 1, which is not reproduced here, shows deflections requiring twice as much time for their completion—i.e., 0.02 second. (14)

(8) Notwithstanding this important difference in deflection time, Figs. 1 and 2 show EKG.'s whose forms agree in all details, and whose various peaks have the same absolute heights within the errors of adjustment—i.e., within a few tenths of a scale division.

(9) We are now justified in making the following conclusion. If the movements of the string could be made 10 or 100 times faster, the sensitiveness remaining the same, or even if, theoretically spoken, an instrument were available with an infinitely small deflection time, the form and dimensions of the recorded EKG.'s would not be thereby perceptibly changed.

(15)

Fig. 1.—Br. Lead I. Magnification of the movements of the string 660. Abscissa 1 scale division = 0.02 sec. Ordinate 1 scale division = 10^{-4} volts.

Fig. 2.—Same person and same lead as in Fig. 1. Magnification of the movements of the string about 1200. Value of abscissae and ordinates as in Fig. 1.

(10) On the other hand, if the movements of the string are sufficiently retarded, the form of the EKG. will also be altered. This is illustrated by Fig. 4. This reproduces the EKG. of the same person taken under the same conditions. The sensitiveness of the galvanometer is, just as in Figs. 1 and 2, accurately regulated so that a deflection of the image of the string of 1 mm. corresponds to 10^{-4} volts. But the string moves much more slowly, and we now see that all the peaks of the EKG. are reduced in size, whilst the ratio of the peaks to one another is markedly changed.

(11) Allow me to make a comparison here of the requirements to be demanded of the galvanometer and the accommodation of the eye. A definite adjustment of the eye corresponds to a definite (16) distance from an object that must be focused sharply. Therefore two objects at different distances from the eye can not form absolutely sharp images at the same time, but the physician knows that if the image of an object at 6 metres distance is sharp, the diffusion circles caused by points farther removed are so small that they may be neglected. The accommodation for infinite distance does not differ practically from the accommodation for 6 metres. In the same way we may say that an EKG. recorded with a galvanometer, of which the deflection time is about 0.01 second, does not differ practically from the same EKG. recorded with a galvanometer whose (17) deflection time is infinitely small.

(12) Within these limits the form of the EKG. is independent of the instrument used, and every EKG, when and where it may have been recorded, is immediately comparable with every other EKG.

(13) A parallel can be drawn between image formation in the eye and the recording of the EKG. (18) in still another direction. Suppose that the accommodation of the eye is faultless in a certain case. Even then the retinal image will not be absolutely sharp and exact. The errors of spherical and chromatic aberration, and of defective centration inherent in all optical systems are also found in the eye; and, moreover, the diffraction of the light rays suffices to exclude the formation of an exact image. In the same way there are sources of inaccuracy in the recording of the EKG.; (19) their influence may be decreased, it is true, but can never be completely avoided. I have here

in mind not so much the dexterity of the observer, who must regulate the tension of the string exactly, so that the curves may be recorded in absolute measure, as the unavoidable changes in resistance which the human body offers to the electrical current.

(14) We must make a distinction between the real and the apparent resistance in measuring the resistance of the body. The real resistance is measured by means of an alternating current, in the same way as the resistance of an electrolyte is determined, whereas the apparent resistance is found by making use of a constant current. Under certain conditions the apparent resistance can have a value of more than twice as much as the real resistance, and the question arises, which resistance are we to take into account when regulating the tension of the string. For example, we wish that a deflection of the image of the string of 1 cm. shall exactly represent 1 millivolt, when the human body is in the galvanometer circuit. If the body resistance is large, the string must be relaxed a little; if on the other hand the body resistance is small, the string must be stretched somewhat more tightly, all other conditions remaining equal.

(15) In case the difference between the apparent and the real resistance of the body reaches an important value, the sharp peak *R* of short duration will be too high in relation to the slow wave *T*, this being an error which is just the opposite of the error produced by a slow instrument.

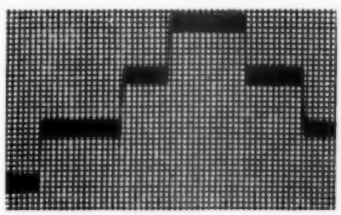

Fig. 3.—Control curve corresponding to Fig. 2. Abscissa 1 scale division = 0.02 sec. Ordinate 1 scale division = 10^{-4} volts.

Fig. 4.—Same condition as in Fig. 1, with the exception that an extra resistance of 100,000 ohms has been introduced into the galvanometer circuit.

(16) How can we best reduce these errors and how large is the error remaining finally? In the first place we must make the resistance of the body as small as possible by putting those parts from which the current is lead off, the hands and the foot, deeply into a strong salt solution. Under these conditions the difference between real and apparent resistance is also reduced to a minimum. Further we must use an instrument with a thin string, thus with a comparatively large internal resistance, so that the variations of the body resistance will only have a small influence upon the current strength.

(17) In regulating the tension of the string we can make use of a rheostat resistance, the value of which is the mean of the real and the apparent body resistance. The difference between these two values need not be larger than 400 ohms under the above mentioned favourable conditions, so that the deviation from the mean will not be more than 200 ohms. If we take the resistance of the string to be 10,000 ohms the maximum error in the dimensions of the curves is 2 per cent.

(18) These errors are much less in reality, because peak *R* is not recorded in an infinitely short time and peak *T* not in an infinitely long time. Moreover, an error of 2 per cent. in the absolute values of the potential difference is so small that it may as a rule be neglected in physiological as well as in clinical investigations. The error would be 0.4 mm. for a large peak *R* of 20 mm. height, and only 0.1 mm. for a large peak *T* of 5 mm.

(19) Although we cannot attain an absolute accuracy at present, yet we may consider an EKG., when properly recorded, the exact expression of the changes in potential difference, just as we may consider the retinal image an exact reproduction of the object observed.

The Form of the Curve in Connexion with the Different Leads.

(20) It is desirable to apply three leads in electrocardiographic investigations: (1) from the two hands, across the body; (2) from the right hand and left foot, obliquely; (3) from the left hand and left foot, lengthwise. The galvanometer must be connected to the human body in such a way that the image of the string is deflected upwards when the base or the right half of the heart is negative in respect to the apex or the left half.

(21) Each of the three leads produces an EKG. of special form so that each has its own peculiar value. Lead I has the advantage of being applied easily in the clinic. The curve obtained is of especial value in judging peak *T* and certain atypical EKG.'s. With lead II the peaks are usually larger. Especially the auricular peak *P* is clearly shown and may therefore be best studied with this lead. In judging the wave *T* in cases of cardiac insufficiency lead II has the same importance as lead I. On the other hand, many atypical forms of the EKG. can be studied better with lead I than with lead II.

(22) Lead III is most suited for diagnosing hypertrophy of the right or left ventricle. Atypical EKG.'s are also well shown by lead III. On the other hand, the wave *T* obtained by this lead is of no value in drawing conclusions as to the functioning of the heart. T_{III} has different heights in normal EKG.'s, is often negative, and often practically absent.

The Connexion between the Forms Obtained with the Three Leads.

(23) From the nature of the case there must be a connexion between the curves obtained by the three different leads from the same person. If two forms are known, the third may be calculated from them. The difference between the electrical tensions of leads I and II must be equal to the electrical tension of lead III. This may be formulated: lead II − lead I = lead III. The formula holds good only in case the current which is flowing through the galvanometer exercises no influence upon the tensions present at the extremities. As a matter of fact, this influence is so small that we may neglect it without making a perceptible error. For the total resistance of the circuit containing the galvanometer and the human body is very large when compared to the resistance of the heart muscle. We may therefore assume *a priori* that the formula is exact.

(24) We might attempt to make use of this formula when examining patients, for the sake of simplicity contenting ourselves with the recording of two leads. The curve of the third lead would then be constructed from the other two. Although the construction seems to be easy, yet its execution gives difficulties, the main cause of which lies in the fact that the peaks, which we generally indicate by means of the same letters, do not occur in identical phases of a heart's revolution. It is possible to show this in various ways—e.g., by comparing the distances between the peaks *P*, *R*, and *T* in the curves obtained by lead I with the corresponding distances in the curves obtained by lead II and lead III. It then appears that the peaks mentioned stand closer to one another in lead I.

(25) A superficial examination of certain curves—for example, those of Fl. (Figs. 5, 6, 7), in which the three leads exhibit fairly large differences—might appear to favour a doubt as to the practical validity of the formula. But it is not difficult to show that the rule is also in accordance with the facts.

(26) The simplest way to do so would be to record two or three curves by the various leads immediately under one another. Two or three large string galvanometers should be used and the images of the three strings should be projected by means of mirrors upon the same photographic plate. It seems to me that the use of a small model of the galvanometer, in which two or more strings are placed in the same magnetic field, or the use of an oscillograph containing a number of mirrors, is not suited to this purpose, because these instruments do not reproduce the form of the EKG. accurately, whereas it is especially desirable in this case to have the form as exact as possible.

(27) Another method has been applied by Fahr and de Waart. They have recorded the EKG., together with the heart sounds, according to the method of Kahn. The EKG.'s obtained by the three leads were taken from the same person under exactly the same conditions, the curve of the heart sounds being combined with each EKG. By this means a fixed point falling always in the same phase of a cardiac cycle could be marked, and corresponding phases in the curves of the various leads could be accurately identified.

(28) The investigation of Fahr and de Waart shows that the ventricle EG. when obtained by leads II and III begins, as a rule, a little earlier than when obtained by lead I, whereas the summit of R_I is reached a little earlier than the summits of R_{II} and R_{III}. If we make use of these data it is not difficult to show the validity of the formula, even in complicated forms of the EKG. To illustrate this we have reproduced in Fig. 8 *QRS*-group of the EKG.'s of Fl. shown in Figs. 5, 6, and 7. The three curves are constructed in the same system of coördinates. The abscissae

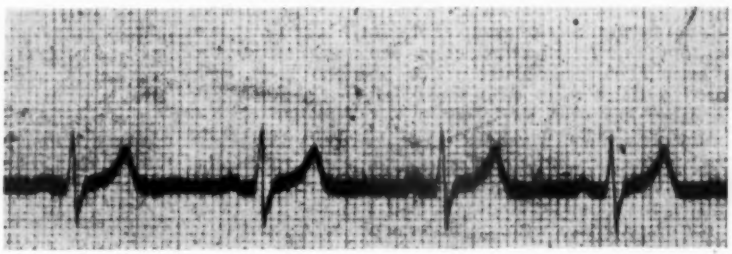

Fig. 5.—Fl. Lead I. Abscissa 1 scale division = 0.04 sec. Ordinate 1 scale division = 10^{-4} volts.*

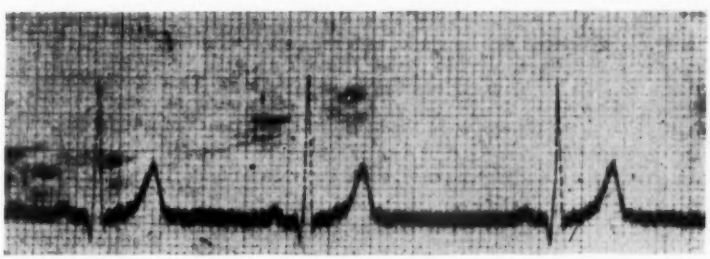

Fig. 6.—Id. Lead II.

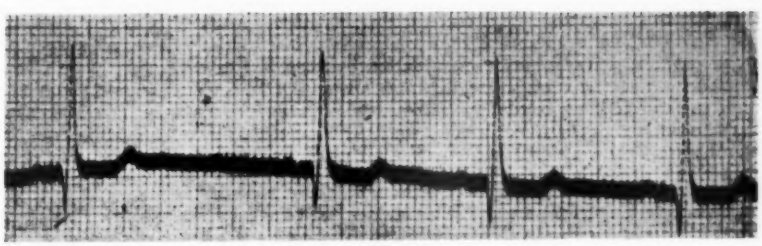

Fig. 7.—Id. Lead III.

have been increased 4 times more than the ordinates, so that the whole figure has been stretched in the direction of the abscissae; one scale division of the abscissae represents 0.01 sec., one scale division of the ordinates 10^{-4} volts. Each vertical line corresponds to one phase of a cardiac cycle and the formula lead II − lead I = lead III holds good for every phase. We see that the peaks Q_{II} and Q_{III} begin 0.01 sec. before R_I and fall in the anacrotic branch of this peak. The whole peak S_I falls in the catacrotic branch of R_{II} and R_{III}, whilst the whole of peak S_{II} only corresponds with the anacrotic branch of S_I.

*If not otherwise mentioned, the abscissae and the ordinates in all figures have the same value as in Fig. 5.

(29) Similar constructions can be made, as a rule, without great difficulty and can be applied to peaks *P* and *T* as well, but it is often difficult to attain a great degree of accuracy. This is especially true when the height of a small peak must be determined from the difference in the heights of two peaks of nearly the same size. If T_I and T_{II} are of approximately the same height, as is often the case, then the value of T_{III}, calculated by means of the formula, is very inexact.

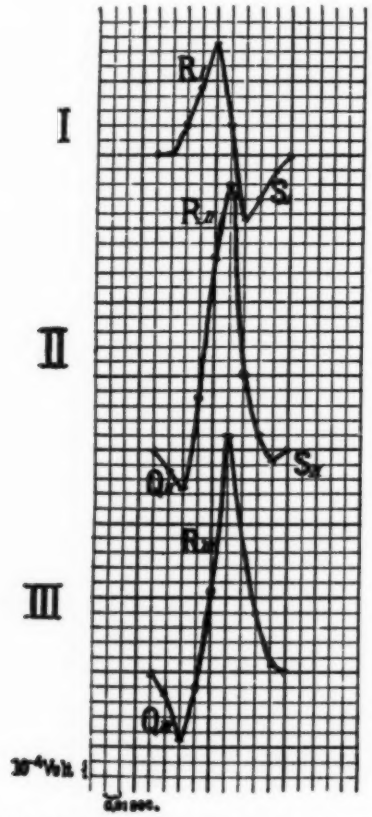

Fig. 8.—Construction of the QRS group in the three leads. Each ordinate represents identical phases of a cardiac cycle. Abscissa 1 scale division = 0.01 sec. Ordinate 1 scale division = 10^{-4} volts.

(30) It is thus evident that we should not content ourselves with taking only two leads, for the dimensions of a curve constructed by calculation can be determined not only more simply but also more accurately by directly recording it by the third lead. The great advantage of a construction as in Fig. 8 is the better insight obtained into the *QRS*-group.

QRS-group.

(31) This group deserves a closer examination because it forms the beginning of the ventricular EG. and shows the path by which the excitation wave is conducted from the auricles to all parts of the ventricular wall. This group takes very different forms in normal hearts, so that it is sometimes impossible to point out the peaks *Q*, *R*, and *S*. The examination of even a small number of curves will show that the inconstant peak *Q* fails in some EKG.'s, the inconstant peak *S* fails in others. In some EKG.'s *S* is as large as *R*, and in some others both *Q* and *S* fail so that only *R* remains.

(32) We reproduce here an EKG. of Wi. (Fig. 9), who possesses a normal heart. The curves obtained by leads I and II show large peaks *R*, as is often the case with normal hearts. The curve reproduced above and taken by lead I exhibits an entirely different form of the *QRS*-group. This group is here represented by a slight elevation, about as high as *P* and about one-third as high as *T*. On closer examination it is seen to consist of two small upwardly directed peaks, placed close beside one another. It is impossible to say whether they are *Q*, *R*, or *S*. The same is true of many normal as well as of many pathological curves taken with lead III. A single example of such a $(QRS)_{III}$-group may suffice (Fig. 10).

(33) We have here again two upwardly and two downwardly directed peaks, no one of which may possibly be distinguished as *Q*, *R*, or *S*. T_{III} is negative in the EKG. of this person.

(34) The explanation of the ventricular EG. is based upon this great variability in the *QRS*-group. As soon as the excitation wave has reached the ventricular walls through the atrio-ventricular bundle and the Purkinje fibres, the ventricular musculature begins to contract in many places at the same or nearly the same time. The atrio-ventricular bundle, according to the investigations of Aschoff-Tawara, consists of a system of muscle fibres, well likened to a tree with its branches. From their origin in the auricular septum to their arborisation in the walls of the lower chambers of the heart, these fibres are separated from the rest of the heart muscles by connective tissue. The bundle does not enter into connexion with the heart musculature excepting with its end ramifications, which are spread over a large surface in the walls of the ventricles, fusing with them at many points. The great variety of forms of the *QRS*-group is caused by small variations in the architecture and the conductivity of the bundle and the Purkinje fibres, so that the excitation wave in some hearts reaches the right side 0.01 second sooner than the left, or the base sooner than the apex, whereas the opposite sequence occurs in other hearts.

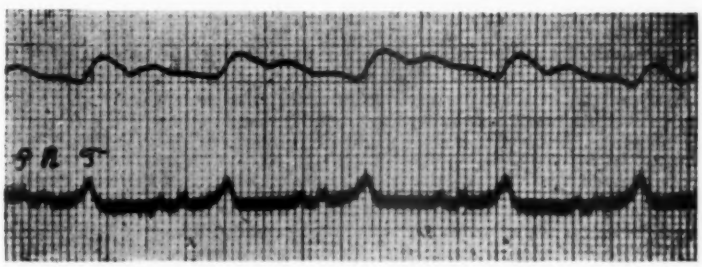

Fig. 9.—Wi. Lead I. A remarkable QRS group.

(35) The contraction wave spreads over the whole ventricular musculature within a few hundredths of a second, and all changes in potential disappear temporarily. This happens in the time between group *QRS* and the beginning of the *T* wave.

(36) The potential difference indicated by the galvanometer in any given moment is the resultant of the joint potential differences existing at that moment between the various parts of the heart. If we consider the phenomena from this point of view we may agree with Selenin, who regards the EKG. as the algebraic sum of the changes in potential, caused by the contraction of the right and left chambers. We also may agree with Eppinger and Rothberger, who assume that the EKG. is the resultant of the forces in the constrictor fibres and in the longitudinal fibres of the heart musculature. Neither is there any objection to the idea of the EKG. being the expression of the antagonism between base and apex. For we may divide the heart into two arbitrary parts, and the EKG. will always be the resultant of the changes in potential differences in these parts. The curve must represent, under all circumstances and in every moment, the algebraic sum of all the potential differences which at that moment are developed in the heart.

Peak U.

(37) We must now point out two peculiarities of the EKG. which up to the present have been considered worthy of only slight consideration. In the first place, the existence of a wave, which

comes after the *T* wave and may have a considerable height in pathological cases. This wave U is also present in the curves from normal hearts, but in these cases it is very low as a rule. Lewis estimates that wave U is present in three-quarters of all EKG.'s taken by lead II. After an examination of my collection of EKG.'s I also conclude that the presence of peak U is very common, at least in half of all persons.

(38) Figs. 11 and 12 are illustrations of EKG.'s with fairly high peaks U, obtained from persons with normal hearts. Fig. 11 is from a sportsman, aged 37 years, with a strong musculature; Fig. 12 is from a well-built young man, 19 years of age, of somewhat more than the average height. Wave U reaches a value of about 0.5×10^{-4} volts in both curves, but is not of equal height in all heart beats. This wave must be regarded as an inconstant one.

(39) The significance of it and the reason for its inconstancy are for the present not known with surety. The method of recording the heart sounds, together with the EKG., has shown that the second sound is produced almost immediately after peak *T* is finished. The end of U lies after the second sound, so that there is no doubt that wave U fails wholly or partly in that phase of the cardiac cycle which follows the closing of the semilunar valves. The heart is in diastole after the closing of these valves. But the heart muscle, which does not begin to contract at all points at the same time, does not relax at all points at the same time either. A large part of the heart can have already relaxed, so that the pressure in the heart's cavities may be practically diminished to zero, and at the same time a few fibres may still be in the condition of contraction. Peak U will not have reached its end until these latter fibres are fully relaxed.

The Interval between Auricular and Ventricular Contraction.

(40) In the second place, we wish to direct attention to the position of the galvanometer in the interval between *P* and the *QRS*-group. In many cases the string takes the same position here as that held during diastole, so that a horizontal line is recorded at the level of the line of diastole. But in many other cases there is a perceptible difference in the positions of the lines. We wish to illustrate this with only one example (Fig. 13). We see in this figure that the curve between *P* and the *QRS*-group lies about 1 mm.—i.e., 10^{-4} volts lower than during diastole. To what must this be ascribed?

(41) Is the conduction through the atrio-ventricular bundle accompanied in this case by the development of a perceptible electrical current, or is a potential difference developed even during the diastole? Have we perhaps to consider a generation of electricity effected by the same causes that produce peak U? For the present we shall not choose between the possibilities mentioned, but shall content ourselves with the information that curves like Fig. 13 are often obtained from persons with normal hearts.

The Influence of the Position of the Heart.

(42) The position of the heart influences the form of the EKG. In electrocardiographic investigations we are chiefly interested, however, in getting a better insight into the *functioning* of the heart and it is easy to understand that a difficulty must arise in drawing conclusions as to this point from the form of the curve, if a displacement of the organ is sufficient in itself to produce a change in the EKG.

(43) The difficulty is best solved by making ourselves accurately acquainted with the effects of a shifting and a rotating of the heart. The easiest and at the same time the most striking example of the influence of the position of the heart upon the form of the EKG. is offered by a case of situs inversus viscerum. Waller was the first to investigate such a case, and since then a number of other cases have been published. I permit myself here, moreover, to publish a curve taken by Dr. Fahr from a boy aged 7 years by lead I. We see immediately that the peaks of Fig. 14 are exactly the reverse of a normal EKG. If we interchange the conducting wires to right and left hands we obtain a curve which cannot be distinguished from a normal EKG., as is shown in Fig. 15.

(44) Whereas the EKG, in situs inversus may be very easily explained and gives no cause for a more extended discussion, other cases of change in position of the heart are accompanied by more complex phenomena, and these require a more detailed analysis for their understanding. We

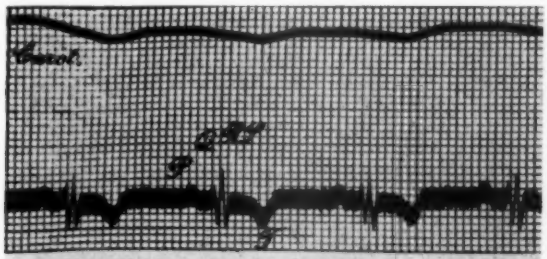

Fig. 10.

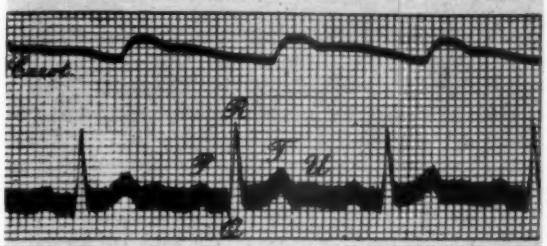

Fig. 11.

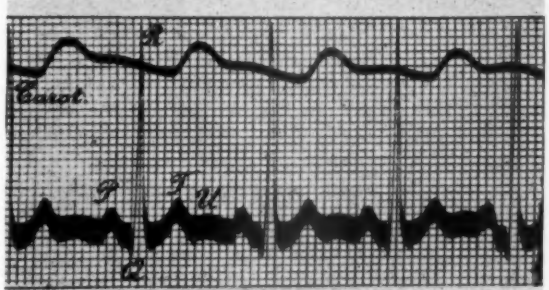

Fig. 12.

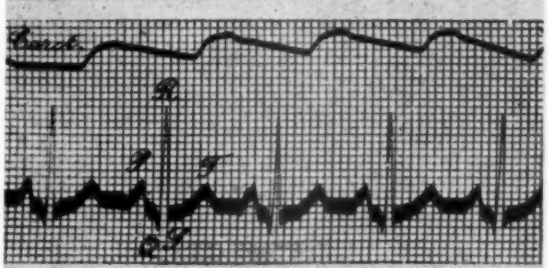

Fig. 13.

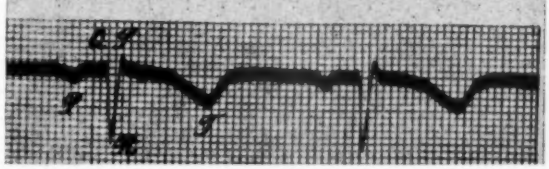

Fig. 14.

Fig. 10.—Jo. Lead III. A remarkable QRS group.

Fig. 11.—Br. Lead II. Wave U visible.

Fig. 12.—Sch. Lead II. Wave U visible.

Fig. 13.—Em. Lead II. The level of the curve between P and Q is different from the level during diastole.

Fig. 14.—Situs inversus viscerum. Lead I. Abscissa 1 scale division = 0.02 sec. Ordinate 1 scale division = 10^{-4} volts.

shall here discuss in particular the deviations in the position of the heart which accompany the movements of respiration.

(45) Movements of the diaphragm participate to a greater or less extent in the respiratory movements of women as well as of men, and the heart must be displaced in the thorax whenever the diaphragm takes a higher or a lower position.

(46) Even before the construction of the string galvanometer Samojloff demonstrated to his classes that increased respiratory movements had a perceptible influence upon the oscillations of the mercury meniscus of the capillary electrometer connected with the human body. By lead I the oscillations became larger in the phase of expiration and smaller in the phase of inspiration. Many other investigators have examined the same phenomena. In our description we shall make use of curves which were taken with the help of Vaandrager and later with the help of Fahr and de Waart. In Figs. 16 and 17 we see pieces of a curve which was taken from Bl. by lead I. The upper curve is the pneumogram.

(47) The pneumogram was obtained by recording the movements of a Hutchinson spirometer into which the person under investigation breathed. A movement of the recording lever upwards corresponds to an inspiration, a movement downwards to an expiration. Each scale division of the ordinates represents 250 c.c. inspired or expired air; the amplitude of the oscillations of the pneumogram varies between 11.3 and 11.5 scale divisions—i.e., 2.8 to 2.9 litres. The vital capacity of the person amounted to 4.4 litres; thus the respiratory movements during the recording of the curves were about two-thirds of the vital capacity. These movements were sufficient for our purpose, and at the same time the subject could execute them with ease.

(48) The pneumogram reproduces the changes in the volume of the thorax without practically any delay. This may be shown by means of the trembling of the subject at the height of inspiration. The effect of this trembling caused by the strong muscle contractions necessary for the ex-, cessive movements of inspiration is to be seen clearly in the EKG. As soon as the inspiration is ended the oscillations in the EKG. disappear, for the muscles relax and passive expiration begins. We may say that the end of the trembling coincides with the top of the wave of the pneumogram, if we neglect small differences in time—as, for example, differences of five scale divisions, or 0.2 second.

(49) Apart from the form of the EKG. and the height of its peaks there is a striking variation in the heart's frequency during the movements of respiration. I should like to point out that the maxima and minima of the heart's frequency do not correspond with the crests and troughs of the pneumogram. For instance, the longest pause in the heart's action is found at the end of Fig. 16, the shortest pause at the beginning of the same figure, whereas the volume of the lungs is about the same in both places. The volume of the lungs is also the same at the beginning and at the end of Fig. 17, whereas the heart's frequency differs markedly in these places.

(50) The explanation of this phenomenon is generally known; during very slow and deep respiratory movements the gases of the blood change sufficiently to influence the tonus of the vagi, especially at their origin in the medulla. As the maximum and minimum of vagus tonus do not correspond to the extreme positions of inspiration and expiration, the time of the greatest and the time of the least frequency of the heart will also be displaced in respect to these positions. This displacement varies largely from person to person. We have mentioned this circumstance here because it must be taken into account whenever the influence of the respiratory movements upon the form of the EKG. is discussed. For we know that the form and magnitude of peak *P* as well as of wave *T* can be materially changed by the action of the vagi.

(51) In Figs. 18 and 19 we reproduce two pieces of a curve, which was taken from the same person by lead III. The influence of the vagi upon the form of peak *P* is shown very clearly in these figures. After long heart pauses *P* is small, sometimes diphasic, other times totally negative, whereas *P* has its normal form and magnitude after small pauses—i.e., with increased frequency and decreased vagus tonus.

(52) The above mentioned facts make it evident that we meet with a difficulty when estimating the influence upon the form of the EKG. of a displacement of the heart caused by the respiratory movements. Peak *P* is influenced by two factors, and it is not easy to separate them from one another nor to estimate the magnitude of each alone. We shall therefore concentrate our attention upon some other peaks of the curve.

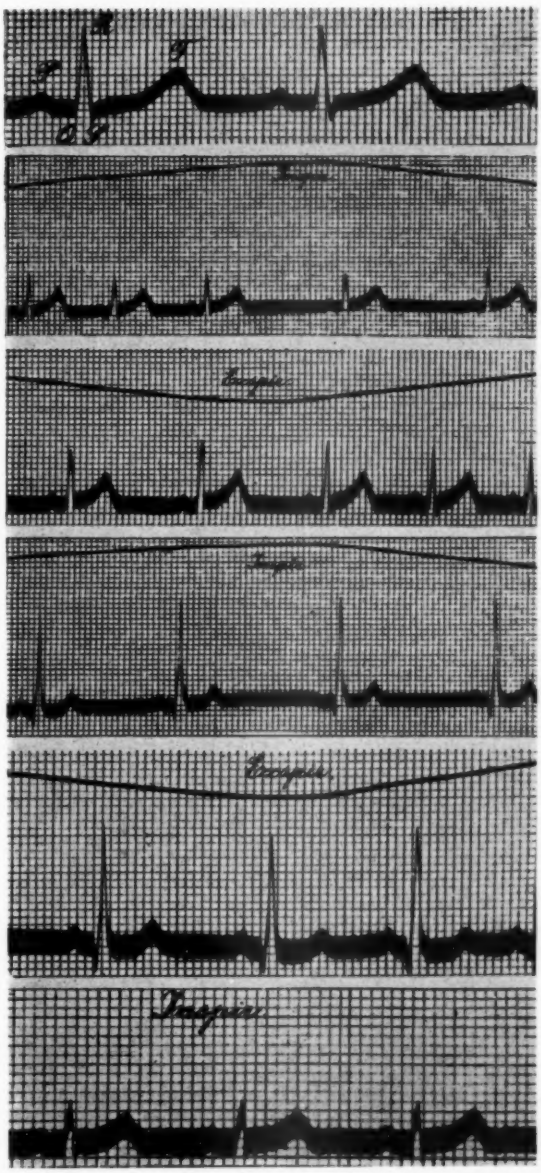

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 20.

Fig. 15.—Id. The connexions of the electrodes reversed.

Fig. 16.—Bl. Lead I during inspiration.

Fig. 17.—Id. Lead I during expiration.

Fig. 18.—Id. Lead III during inspiration.

Fig. 19.—Id. Lead III during expiration.

Fig. 20. Bak. Lead I during inspiration.

(53) We see from Figs. 16-19 that the peaks of lead I are larger during expiration, whereas by lead III they become smaller during the same phase of respiration. If we consider these facts in connexion with the formula, lead III = lead II—lead I, we must conclude that the changes in the peaks of lead II will be very slight. And this is, in fact, the case with Bl. and with the majority of the persons investigated. The practical result of this is that we must apply this lead when examining a patient if we wish to investigate the form of the EKG. as free from the influence of the respiratory movements as possible.

(54) Further, we wish to remark in the above figures that peaks *R* and *T* are both decreased or both increased in the same degree as well in lead I as lead III during the various phases of breathing. Thus the relative heights of the peaks remain the same, so that the curve retains its form.

(55) But similar changes are not observed in all persons. In some persons we find differences which upon superficial examination seem to be wholly irregular and thereby puzzling. We shall first describe some of these differences separately, and shall then try to unite them all under one point of view.

(56) In the first place we shall direct attention to the fact that the changes which peak *R* undergoes are, in many cases, materially larger than the corresponding changes in *T*. Figs. 20, 21, 22, and 23 are examples of this. Figs. 20 and 21 reproduce the EKG. of Bak. by lead I. Two pieces have been cut out of the original curve in such a way that Fig. 20 renders that portion of the record taken in inspiratory position, and Fig. 21 renders that portion taken in expiratory position. In this latter figure we see that the enlargement of peak *R* is materially greater than that of the *T* wave, whereby the curve receives quite another shape.

(57) An even greater disparity is brought to light in Figs. 22 and 23, which were obtained from Wi. Here we have again two pieces of a curve taken by lead I; Fig. 22 corresponds with the inspiratory, Fig. 23 with the expiratory position of the person under investigation. In the first position *R* is larger than *T*, in the second the relation is reversed.

(58) Another difference found is that a peak which becomes larger in lead I also increases in lead III. This phenomenon occurs whenever the peaks have opposite directions in the above-mentioned leads. Figs. 24 and 25 furnish an example of this. They reproduce the EKG. in lead III of the same person whose EKG.'s in lead I were shown in Figs. 20 and 21. Wave *T* is negative in lead III and we see that T_{III} has increased as well as T_I. Phenomena similar to those described above are to be observed in the *QRS*-group.

(59) But we shall now try to explain the apparently so irregular effects of the respiratory movements upon the form of the EKG. In the first place we exclude the possibility that such potential differences as are shown by our curves might be due to accidental causes—as, for instance, the muscle contractions necessary for the respiratory movements, or to a displacement of the electrodes. For if such secondary causes should exercise an influence synchronous with the movements of respiration, then the curved line which reproduces a series of EKG.'s would have to rise and fall in the same rhythm as the movements of breathing. But we see that the line drawn through the heart pauses does not show such a rhythm. On the contrary, it has in many curves an absolutely straight course, as may easily be seen with the aid of the system of square millimeters.

(60) As a second possibility to be taken into account we may mention the influence of a changing body resistance, a hypothesis pointed out by Samojloff. The electrical resistance of the organs surrounding the heart must show during breathing rhythmic changes due to changes in the blood and air content of the chest, and these changes in resistance must necessarily influence the heights of the peaks of the electrocardiogram. But this influence is obviously small, for the curve of lead II shows only slight changes as a rule, whereas the changes produced in lead III are as a rule the opposite of those in lead I. Moreover, the fact that the relation between the heights of the peak is often changed cannot be explained by a change in the resistance of the body.

(61) As a third cause we may mention that the manner in which the heart contracts depends upon the pressure in the thorax. The negative interpleural pressure, which increases during inspiration and decreases during expiration, mechanically influences the circulation perceptibly. But this influence is also insufficient to explain the phenomena described.

(62) The difficulties are solved at once, however, if we apply a schema in which the human body is represented by a plane, homogeneous plate in the form of an equilateral triangle, *RLF*. (Fig. 26.)

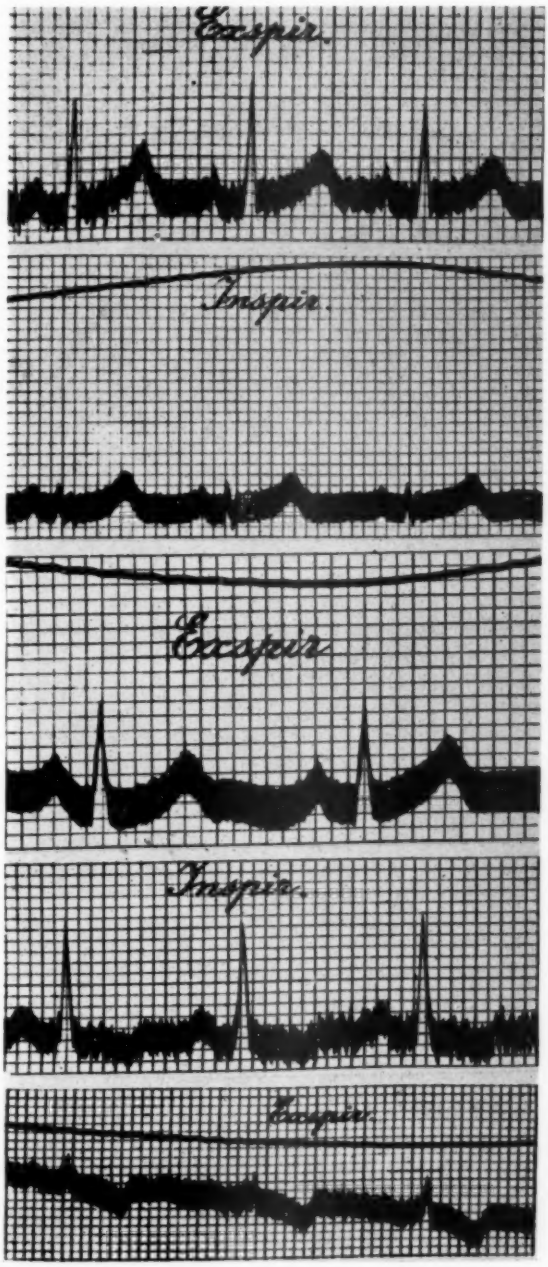

Fig. 21.

Fig. 22

Fig. 23

Fig. 24.

Fig. 25.

Fig. 21.—Id. Lead I during expiration.

Fig. 22.—Wi. Lead I during inspiration.

Fig. 23.—Id. Lead I during expiration.

Fig. 24.—Bak. Lead III during inspiration.

Fig. 25.—Id. Lead III during expiration.

From its corners the current is lead off to the galvanometer. *R* corresponds to the right and *L* to the left arm, while *F* indicates the potential of both feet. Thus, a lead from *R* and *L* corresponds to lead I, from *R* and *F* to lead II, and from *L* and *F* to lead III. A small spot *H* in the middle of the triangle represents the heart. We assume that in a given moment the potential differences in the heart are so distributed that their resultant takes the direction of the arrow drawn in the figure. This direction indicates the direction of the current in the heart. The angle which the arrow forms with the side *RL* is called α. We shall call the angle positive if, when looking towards the front wall of the chest of the patient, the arrow rotates with the hands of the clock; negative if it rotates in the opposite direction.

(63) We now suppose that the EKG. of a patient has a simple form in each of the three leads, so that the peaks R_I, R_{II}, and R_{III} fall in corresponding phases of a cardiac cycle, and $R_{II} - R_I = R_{III}$. If we transfer the values found with the patient to the schema we are able to determine the direction of the potential difference which has been present in the heart during the recording of R.

(64) The EKG. of Bak. in the position of inspiration may be taken as an illustration. In his curves the following values are found for R: $R_I = 3.2$, $R_{II} = 12.5$, $R_{III} = 9.3$ millivolts. In the homogeneous triangular plate with the heart in the centre these values can only be produced by a potential difference, having the direction of the arrow drawn—i.e., that $\alpha = 76°$.

(65) In the position of expiration we find for the same person: $R_I = 9.2$, $R_{II} = 11.2$, $R_{III} = 2.0$; wherefrom we can calculate that under the circumstances $\alpha = 40$. (*Vide* Fig. 27.)

(66) Thus we are able to demonstrate by means of the schema that the heart has rotated in the chest around a sagittal axis during the movement of expiration. The amount of rotation according to the schema is $\alpha_1 - \alpha = -36°$. As we have reason to believe that the real amount of rotation in the body corresponds sufficiently well for practical purposes with the amount of rotation in the schema, we shall hereafter simply consider the direction of the potential difference in the body as parallel to the direction of the arrow in the schema. The way in which α can be calculated from the potential differences recorded need not be explained in detail. It may suffice to state that the calculation is simple and can be executed very quickly.

(67) The schema can show more than the direction of the potential difference only. It also makes it possible for us to compare the values of the potential differences present in the heart itself. To make this clearer we shall introduce a new term, "the manifest potential difference in the heart," and when speaking of certain peaks, *P*, *R*, or *T*, we shall indicate them with the figures *Pm*, *Rm*, or *Tm*. By these figures is meant the manifest values of the peaks.

(68) We define the manifest potential difference in the heart as the amount shown in one of the three leads, when the direction of the current between the points of leading off is parallel to the direction of the potential difference in the heart. We here consider only potential differences in a frontal plane. Suppose that a given potential difference in the heart forms the peaks T_I, T_{II}, and T_{III}, and that the direction of the current in the schema is represented by an arrow parallel to the side RL (see Fig. 28), then T_I is the largest of the three peaks, T_{II} and T_{III} being opposite to one another, and each equal to one half of T_I. In this case $Tm = T_I$.

(69) The manifest value of a peak can always be calculated from the height of the recorded peaks and thus can always be expressed in absolute measure—e.g., in tenths of a millivolt. This calculation is also easily and quickly made. If we apply it to peak *R* of Bak., the dimensions of which have already been given, we find $Rm = 13 \times 10^{-4}$ volts in the position of inspiration, $Rm = 12 \times 10^{-4}$ in the position of expiration.

(70) In another person a rotation of the heart of $-12°$ was brought about by expiration, *Rm* amounting to 27×10^{-4} volts in a position of inspiration and to 26×10^{-4} volts in a position of expiration. In a third person a rotation of $-20°$ was found, whilst *Rm* kept the constant value of 20×10^{-4} volts. In all persons examined till now the rotation takes place in the same direction in expiration, whilst the manifest height of *R* keeps constant within the limits of the errors of the method.

(71) We can investigate the direction and the manifest value for *T* in the same way as for *R*. As is to be expected, *T* is always rotated by the movements of respiration in the same direction as *R*, but the amount of rotation is not exactly the same. Moreover, the manifest value of *T* changes—a fact which points out that other factors besides the rotation of the heart are active in the formation of *T* during respiration. The vagus tonus is one of these factors.

Fig. 26. Fig. 27.

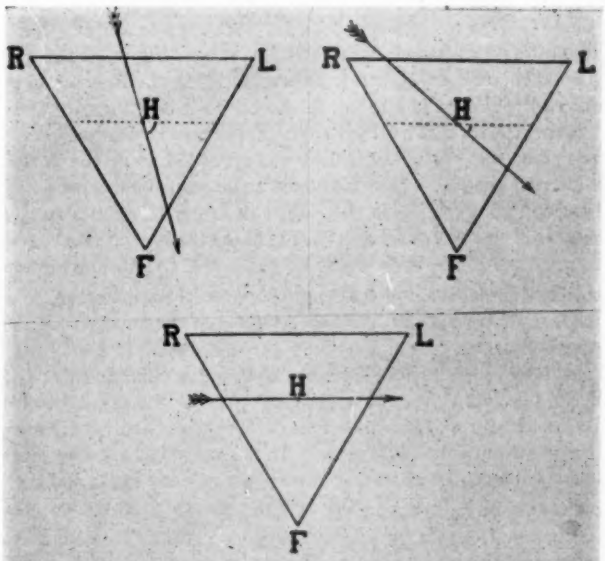

Fig. 28.

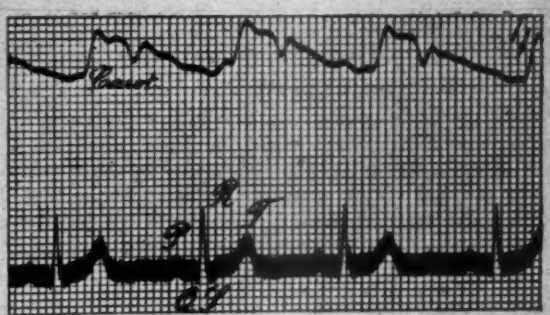

Fig. 29.

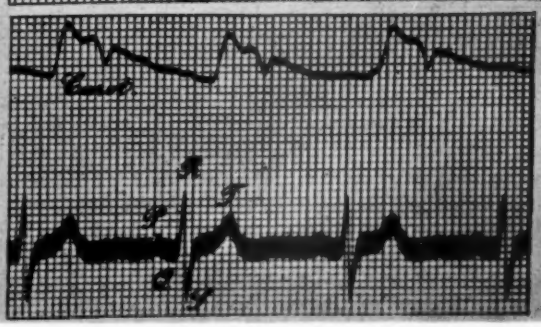

Fig. 30.

Fig. 26.—Schema of the equilateral triangle. *R* corresponds to the right hand, *L* to the left hand, and *F* to both feet. The heart, *H*, is in the centre. The arrow indicates the direction of the potential differences in the heart. α = 76 degrees.

Fig. 27.—Id. α = 40 degrees.

Fig. 28.—Id. α = 0 degrees.

Fig. 29.—Fl. Lead I, patient lying on his left side.

Fig. 30.—Id. with same lead, lying on his right side.

(72) There are good reasons for assuming that the most prominent peaks of the ventricle electrogram, *T* and *R*, are brought about by potential differences localised in approximately frontal planes. This is proved by the behaviour of peak *S* in lead I. When a person lying horizontally upon the left side turns over upon the right side whilst his EKG. is taken, we obtain the striking phenomenon that peak S_I, which is often very small or not present at all in the first position, becomes visible and sometimes reaches a prominent height in the second position. Figs. 29 and 30, both of which were taken by lead I, furnish an illustration of the production of a large peak S_I by the turning of the body on to the right side. Fig. 29, which was recorded while the patient was lying upon the left side, shows a small peak *S*, which may almost be neglected, whereas Fig. 30, which was obtained while the patient was lying upon the right side, shows a peak *S* so large that we are inclined to be astonished that both curves have been obtained from the same person, with the same lead, and, with the exception of the body position, under quite the same circumstances.

(73) The most satisfactory explanation of this, upon superficial examination, so puzzling phenomenon is to be found in the assumption that the heart rotates in the chest about the longitudinal axis of the body, when the body itself turns from the right upon the left side. If there is a potential difference in the heart having a sagittal direction, then a slight rotation of the organ about the longitudinal axis of the body can produce a material change in the height of the corresponding peak, and this will be especially well shown by lead I. If the potential difference has exactly the sagittal direction the peak entirely disappears. It is probable that the large differences in *S* in different persons, as well as its inconstancy, must, as a rule, be explained by this circumstance.

(74) We direct attention to the fact that in Figs. 29 and 30 the peaks *P*, *R*, and *T* undergo hardly any change. If we conclude from the enormous change in *S* that a rotation of the heart has taken place round the long axis of the body, then we must also assume that the peaks *P*, *R*, and *T* are developed in approximately frontal planes, or in other words, in planes making relatively large angles with the sagittal plane, for thereby it can be most easily explained that under these circumstances the changes in these peaks are so small.

(75) The application of the schema of the equilateral triangle accounts for a number of phenomena here treated of. First it is immediately obvious from the schema that a comparatively large displacement of the heart parallel to itself will only slightly affect the heights of the peaks recorded. This explains the fact that the EKG. of many persons is hardly perceptibly changed or is not changed at all during the ordinary movements of breathing. It also explains the fact that with the exception of peak *S*, there are only slight changes produced in the EKG., when the body is turned from one side to the other, although we must expect from the anatomical conditions that the heart has hereby taken a lower position in the chest.

(76) The schema explains easily and satisfactorily how the various peaks of the EKG. are often unequally increased or decreased by a change in the position of the diaphragm, and why the curve of lead II shows slighter changes than the curves of leads I and III. A potential difference which is present in the heart and has a direction which in the one position of the heart makes an angle of 80°, and in the other position an angle of 40°, according to the schema, causes the formation of a peak which remains unchanged in lead II, but which is very much altered in lead I as well as in lead III. If this peak in lead II has the value 9.4×10^{-4} volts in the first position of the heart, it keeps this value in the second position. It increases in lead I from 1.7×10^{-4} volts in the first position to 7.7×10^{-4} volts in the second. It decreases in lead III from 7.7×10^{-4} volts in the first position to 1.7×10^{-4} volts in the second.

(77) It is equally clear why a peak which has the same direction in leads I and III undergoes opposite changes by a displacement of the diaphragm, whereas it is either increased or decreased in both leads if it has a positive value in lead I and a negative in lead III. This is the case when α has a value between $-90°$ and $+30°$.

(78) With the aid of the schema we are in a better position to judge the form of the EKG. under all circumstances. For instance, it is possible therewith to show that an increased frequency of the heart brought about by bodily exercise is always accompanied by a positive rotation of *T* and by an increase in the manifest value of this peak. In myodegeneratio cordis and in many cases of auricular fibrillation *T* is small in all three leads. It need not be said that *Tm* must also be small under these circumstances.

(79) In cases of hypertrophy of the left ventricle we sometimes find the most prominent peak of the *QRS*-group in a direction represented by $\alpha = 40°$, whereas in normal hearts the value of α for this peak lies between $+ 40°$ and $+ 90°$. We see from these figures that the deformation of the EKG. in this heart affection is not, or not chiefly, caused by a change in the position of the heart. The manifest value of the most prominent peak of the *QRS* group in hypertrophy of the left ventricle can reach the enormous value of 26×10^{-4} volts. In hypertrophy of the right heart we obtain, as is well known, EKG.'s of an entirely different form, to which, however, similar considerations may be applied. By means of the schema we can in general differentiate the real changes in the heart's action from the apparent ones, which are only caused by variations in the position of the heart.

(80) Atypical contractions may also be examined with the aid of the schema. They are of great importance in the clinic, and are worthy of a special, detailed discussion, but I must conclude now.

(81) It gives me an especial pleasure to bring to remembrance here that the human EKG. was first recorded by a London physiologist, Augustus D. Waller, who also introduced the term "electrocardiogram" into science; and that Dr. Thomas Lewis, whose extensive researches have been crowned with such exceptional success, was the first man in England who applied electrocardiography to clinical investigations.

(82) The method of electrocardiography is still a young plant. We may reasonably expect that it will continue to bear good fruit.

WHAT IS THE REASON FOR THE VENTRICULAR ARRHYTHMIA IN CASES OF AURICULAR FIBRILLATION?

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THE fact that auricular fibrillation in man is the cause of absolute arrhythmia* of the ventricles has been known since 1909, when it was recognized by Winterberg and Rothberger.[4] However, the precise mechanism by which this arrhythmia is induced remains obscure, and strangely enough very few serious attempts have been made to explain it. Hoffmann[1] was the first to point out that theoretically one would expect auricular fibrillation to be followed by a slightly irregular (or nearly regular) ventricular rhythm, and not by the grossly irregular rhythm which is usually found. He considered auricular fibrillation simply an auricular activity of high frequency which results in a rapid bombardment of the A-V node with impulses. This bombardment is ineffective during the refractory period† but, owing to the fast rate of fibrillation, an impulse may always be expected to reach the node very soon after the end of the refractory period. The consequent ventricular rhythm will be nearly regular since all R-R intervals should be approximately as long as the refractory period of the A-V node.

In extreme tachycardia the ventricles really seem to behave in this way, at least for short periods (see Fig. 3, the period between *C* and *D*). However, the sequence of such short and nearly regular intervals is usually broken now and then by isolated long intervals. With falling ventricular rates long intervals become more frequent and in cases with "normal" rates (60 to 100 per minute) the ventricular rhythm is usually a typical absolute arrhythmia with R-R intervals which may vary between 0.60 and 2.00 seconds in haphazard sequence. It is difficult to understand the mechanism of such extreme arrhythmias and the main difficulty is to explain the formation of the long intervals.

Most authors accept the refractory period of the A-V node as the main factor governing ventricular rate in auricular fibrillation. Reasonably, a longer refractory period should result in longer R-R intervals and vice versa. The duration of this period in man is not known, but it is improbable that it could reach 2.00 seconds and still less probable that it could vary between 0.40 and 2.0 seconds during the tracing of one electrocardiogram. Consequently there must be some other explanation for the occurrence of long intervals.

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*In this paper the term auricular fibrillation will be used for the atrial activity only. The consequent ventricular arrhythmia will be referred to as absolute arrhythmia.

†This term will be used in this paper to indicate the period of refractoriness against the normal stimulus (i.e., the atrial activation impulse). Admittedly, this definition is not very satisfactory in cases of auricular fibrillation. It will be evident from what follows that the imperfections of this definition are of little consequence for the main conclusion in this study.

Some authors avoid this difficulty by speaking about different degrees of block in the atrioventricular node. The only type of block which may cause considerable irregularity and slowing of the ventricular activity is a second-degree (or third-degree) block. The situation in sinus rhythm makes the assumption of such a block (with dropping of ventricular beats) seem a satisfactory explanation for the formation of long R-R intervals. If during sinus rhythm one impulse fails to pass the atrioventricular system, then the ventricles will remain silent for two ordinary R-R intervals, and if this happens frequently, a considerable arrhythmia will result. It is seldom realized, however, that conditions are different during auricular fibrillation. If one fibrillation impulse fails to activate the node, then the next impulse will be there within 0.10 to 0.20 second, and if this one also fails, the third impulse will turn up within the same short interval. Thus the presence of auricular fibrillation per se eliminates the main condition for the formation of long intervals which are characteristic of second-degree block in sinus rhythm.

Some other theories will be mentioned briefly. Many authors assume that fibrillation impulses are of varying strength, and that most of them are too weak to activate the atrioventricular node or that some of them fail to reach the node. Others assume that the incessant bombardment of the node may affect its excitability in an unpredictable way. These theories are of little consequence to the solution of the problem proposed in this paper. Therefore I have refrained from a full discussion of them.

Most authors agree that the mechanism of absolute ventricular arrhythmia due to auricular fibrillation has not been satisfactorily explained. I have concentrated on the question: how are the long R-R intervals of absolute arrhythmia formed? Some observations reported here will contribute to the solution of this question.

METHOD

The method in this investigation was a rather simple one. In cases with auricular fibrillation, long electrocardiogram tracings were recorded (usually about 100 ventricular cycles), and all R-R intervals were measured. Several methods were tried for tabulating the results; the best one proved to be the one illustrated in Fig. 1. It was originally adopted in order to shorten the long electrocardiogram tracings by registering the R-R intervals vertically on a chart in the same order as they occurred in the electrocardiogram. By this method it became possible to observe changes in duration of the individual R-R intervals during periods of changing ventricular rates.

This possibility proved to be of particular interest since most observations of importance were made during such periods of changing rates. The discussion in this paper is concerned mainly with the observations on twenty-four cases in which the falling rate after exercise tachycardia was recorded. In addition, the changes in heart rate were followed in ten cases by making daily electrocardiograms during digitalis treatment. In the cases observed after exercise the electrocardiogram was recorded continuously until the heart rate had reached that before exercise.

The criteria of auricular fibrillation used were: absence of P waves, absence of regular F waves of the type seen in auricular flutter, and presence of ventricular arrhythmia. In most, but not all, cases irregular F waves of the fibrillation type were observed, at least for short periods.

In most cases a direct-writing Cardiotron apparatus was used. Only Lead II or in some cases a chest lead (CR_2) was recorded.

OBSERVATIONS

Current conceptions of absolute arrhythmia made (or should have made) it seem likely that the dots in such charts would be diffusely spread out over the considerable space between maximum and minimum values. A distribution picture corresponding to this expectation was found in twenty of the cases (see Fig. 2). The frequency curve corresponding to such charts was always in a typical way assymmetrical with the summit in the region of shorter intervals and a prolonged slope toward long intervals. Little information of immediate interest could be obtained from a direct study of such charts.

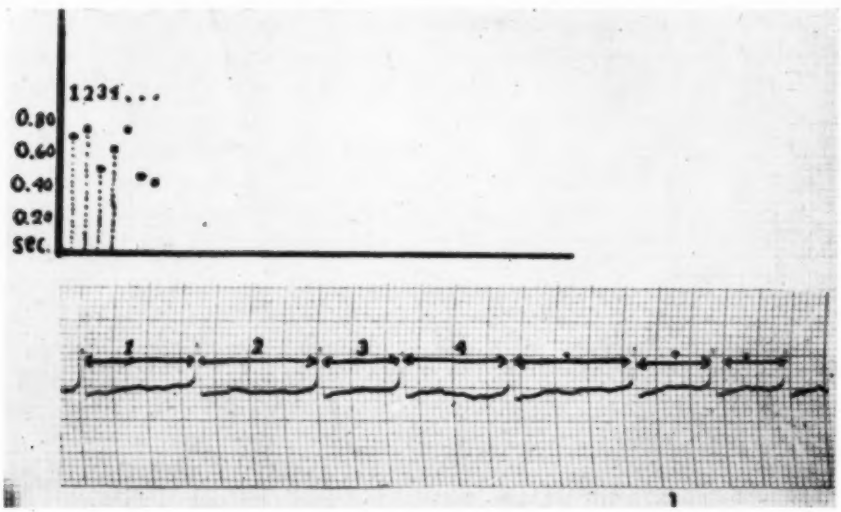

Fig. 1.—The charts to follow have been made in this way: Scale to the left represents 100th of a second.

The charts of the remaining fourteen cases presented a particular feature which will be referred to in the following pages as formation of levels. In the present paper attention will be focused on these cases. It is true that they represent only a minority of the material. I believe, however, that they do not differ essentially from the other cases; the same plan of levels may be present in both groups of cases though not always distinctly demonstrable.

Patient A. W. (Fig. 3) may be used as a convenient starting point for description of the findings in seven of these cases. The patient is a woman of 61 years with mitral stenosis, long-standing auricular fibrillation, and frequent

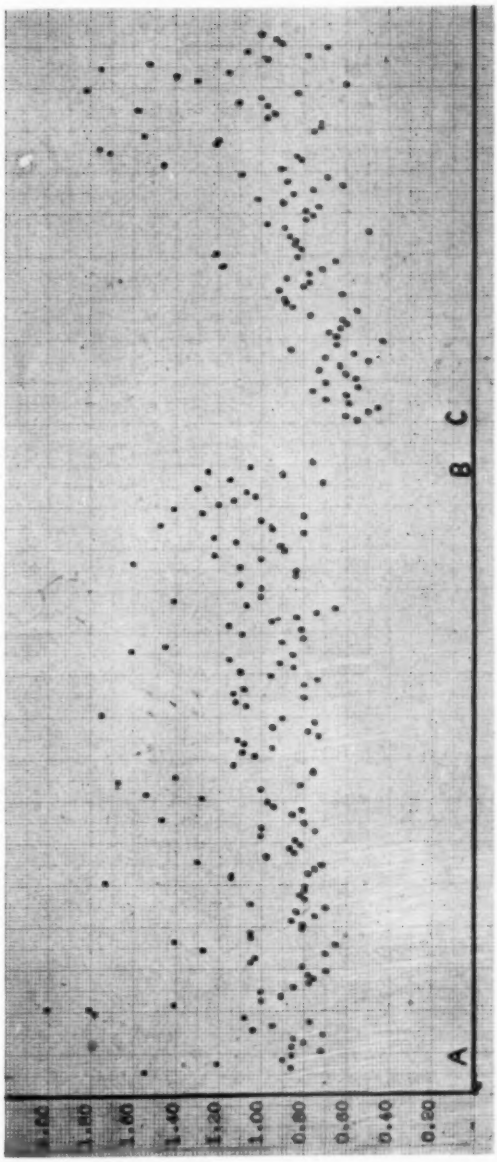

Fig. 2.—Chart of a man of 82 years with arterial hypertension and auricular fibrillation. He had moderate congestive failure and was well digitalized when the chart was made. *A* to *B* was before exercise; between *B* and *C*, moderate exercise; after *C*, post-exercise tachycardia. There was no differentiation of levels. Scale to the left: 100ths of a second. (Patient A. H.)

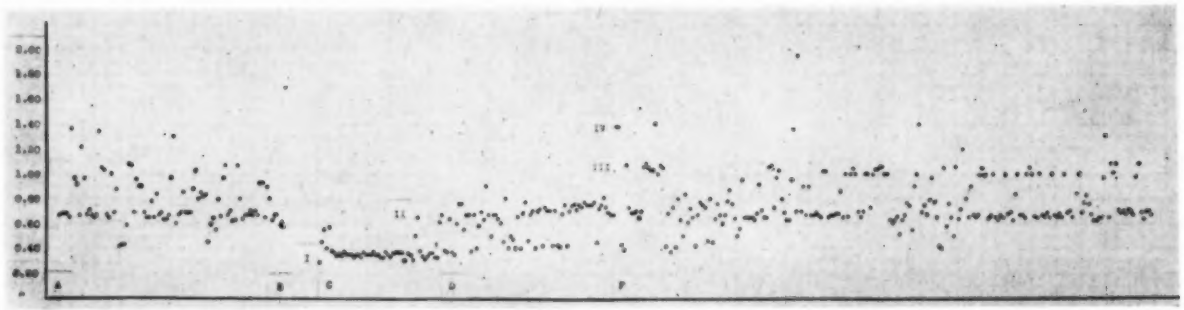

Fig. 3.—Chart of a woman of 61 years with mitral stenosis, moderate congestive failure, and auricular fibrillation. She was digitalized when the chart was made. *A* to *B* was before exercise; *B* to *C*, moderate exercise; after *C*, post-exercise tachycardia. There was distinct differentiation into levels (marked I, II, III, and IV). Scale to the left: 100ths of a second. (Patient A. W.)

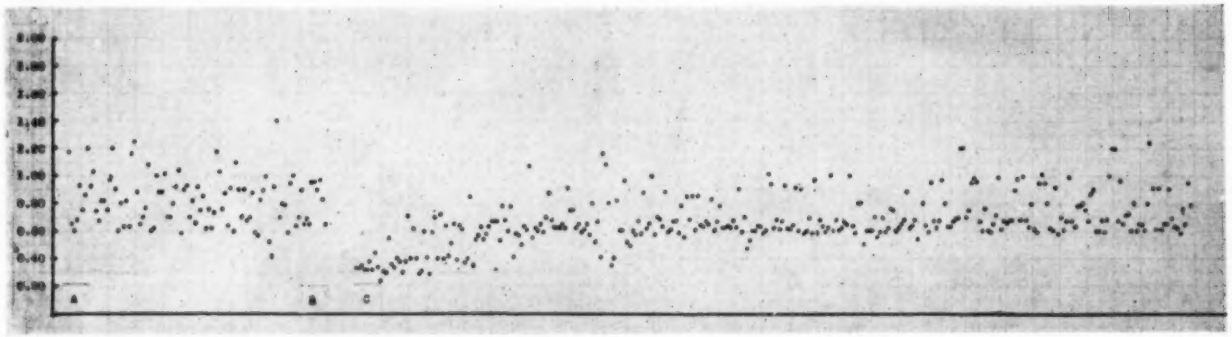

Fig. 4.—Chart of a man of 49 years with auricular fibrillation of unknown etiology (sinus rhythm restored with quinidine about one week later). *A* to *B* was before exercise; *B* to *C*, moderate exercise; after *C*, post-exercise tachycardia. Three levels may be traced after exercise. Scale to the left: 100ths of a second. (Patient E. N.)

episodes of congestive failure. The chart was made at a time when she was in tolerably good general condition. The first part of the chart represents the ventricular activity during rest; the second part (after *C*) represents the tachycardia after very moderate exercise and its slow return to the initial rate.

The first part of the chart shows little of special interest. The dots are spread out rather diffusely between the limits 0.40 to 1.40 seconds but are somewhat more numerous at the level 0.65 to 0.70 second. The picture is thus similar to the one found in the majority of the cases of absolute arrhythmia.

The tachycardia after exercise is expressed by a concentration of the dots at a distinct level at about 0.35 second (Level I). The R-R intervals differ so little in length that the rhythm appears nearly regular. This type of ventricular activity may be regarded as an ideal realization of the theoretical concept of Hoffmann. The moderate irregularity (*in absolute figures*) of the fibrillation itself may well account for the degree of ventricular arrhythmia present.

The return of the ventricular activity to normal would give the impression of a smooth and uneventful transition if it were followed in a simple pulse chart. The chart of intervals shows, however, that this transition is actually stepwise and not smooth.

Level I is at first horizontal, but eventually it shows a slow rise from 0.35 to about 0.40 second. At the same time, dots are seen to concentrate at a new level (Level II), which finally settles at just below 0.70 second. At first these levels coexist but the dots of Level I become fewer and fewer and finally this level disappears completely. Before the complete disappearance of Level I a new level of dots forms at about 1.00 second (Level III) and at the same time a still higher level (Level IV) may be traced just below 1.40 seconds.

At this time the ventricular rate has returned to what it was before exercise. Looking back at the chart of the latter period one realizes that the concentration of dots at 0.65 to 0.70 second corresponds to Level II of the period after exercise, and that traces of the three other levels may also be seen before exercise. Thus exercise has in this case the effect of demonstrating more clearly the existence of levels which were otherwise difficult to discern. In six other cases a similar differentiation of levels was observed after exercise. In most cases it was not quite so distinct as in patient A. W.

Fig. 4 may be regarded as an average specimen. In all the cases the phenomenon was reproducible several times. It may be added that a concentration of the dots at 0.35 to 0.40 second (= Level I) was observed in almost all cases of absolute arrhythmia with high frequency. Such cases, however, in which Level I only was present have not been included in the group of cases "with distinct levels."

I would like to emphasize the rather constant position of these levels in all the cases. Level I was always at 0.35 second as long as it was the only level in extreme postexercise tachycardia; with lower rates it usually settled to 0.40 second. Level II was always found at 0.65 to 0.70 second and Level III at 1.00 second. Level IV was visible only in Patient A. W. (It may be identical with the "ceiling" level to be discussed later on.)

In four of ten cases observed for 1 to 2 weeks during digitalis treatment, similar levels were periodically present (Figs. 5 and 6). In one of these cases (Fig. 5) Levels I and II rose to higher positions (0.50 and 0.90 second) than in all the other cases.

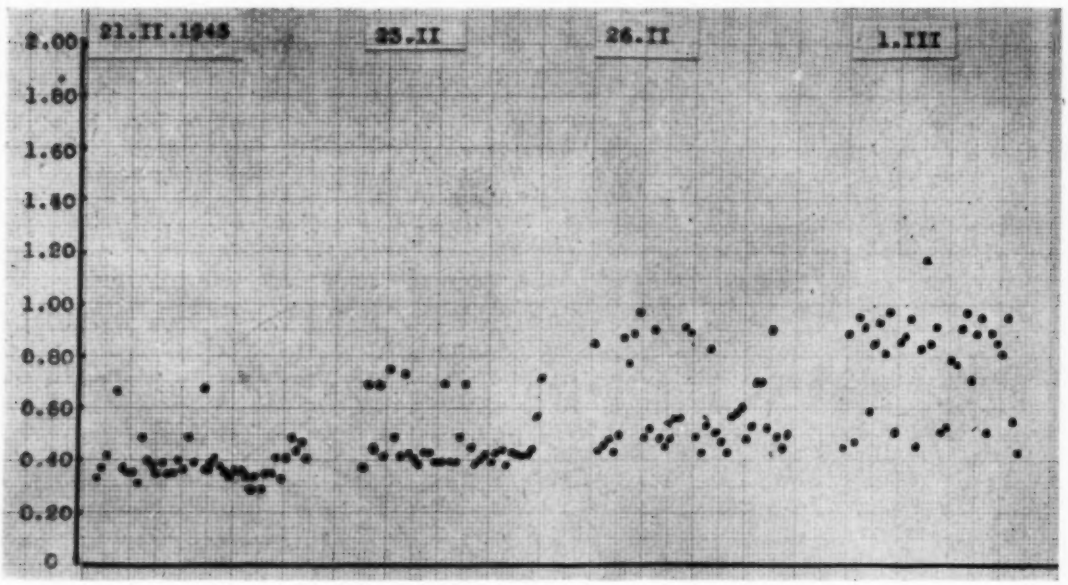

Fig. 5.—Chart of a man of 58 years with coronary sclerosis, admitted with congestive failure and acute auricular fibrillation. The patient received 0.3 Gm. of folium digitalis daily between February 21 and March 1. The chart illustrates the depression of the ventricular rate during this time. Scale to the left: 100ths of a second. (Patient D. M.)

A different type of level was observed in the three remaining cases. In contrast to the ones just described it disappeared during post-exercise tachycardia and reappeared in extreme bradycardia, forming something of a "ceiling" or upper limit at about 1.30 to 1.80 seconds (see Fig. 7). In two of these cases sinus rhythm was restored with quinidine, and both then showed a second-degree atrioventricular block with dropping of ventricular beats. There is no reason to assume that in these cases the condition of the atrioventricular system was essentially different immediately before the return of sinus rhythm. The long intervals forming the ceiling level may be the intervals of intrinsic nodal or ventricular rate during periods of complete atrioventricular block.

DISCUSSION

The observations related above suggest that there is an intrinsic system in the apparent chaos of absolute arrhythmia. If a sufficient number of intervals were measured, the figures obtained in many cases would indicate the presence of fixed levels of frequency, two to four of which could be seen at the same time in the charts. The absolute position of these levels was roughly the same in

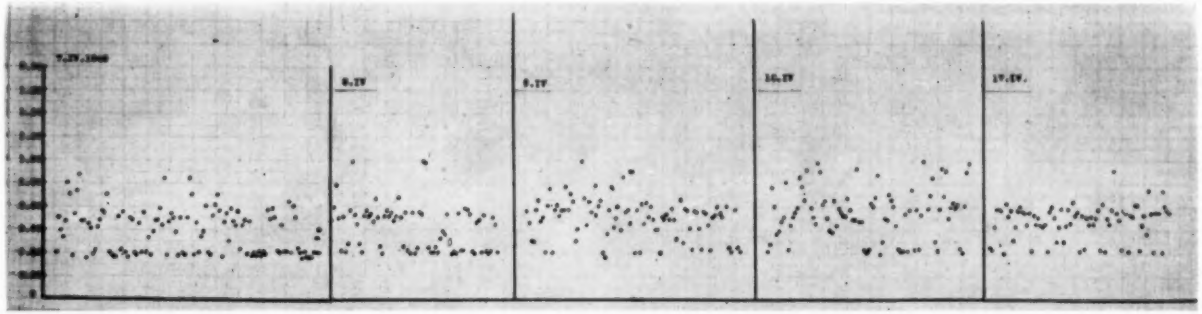

Fig. 6.—Chart of a man of 55 years with mitral stenosis and congestive failure during digitalis treatment. He had long-standing auricular fibrillation. The heart rate fell from 112 per minute (April 7) to 75 per minute (April 10). Scale to the left: 100ths of a second. (Patient B. K.)

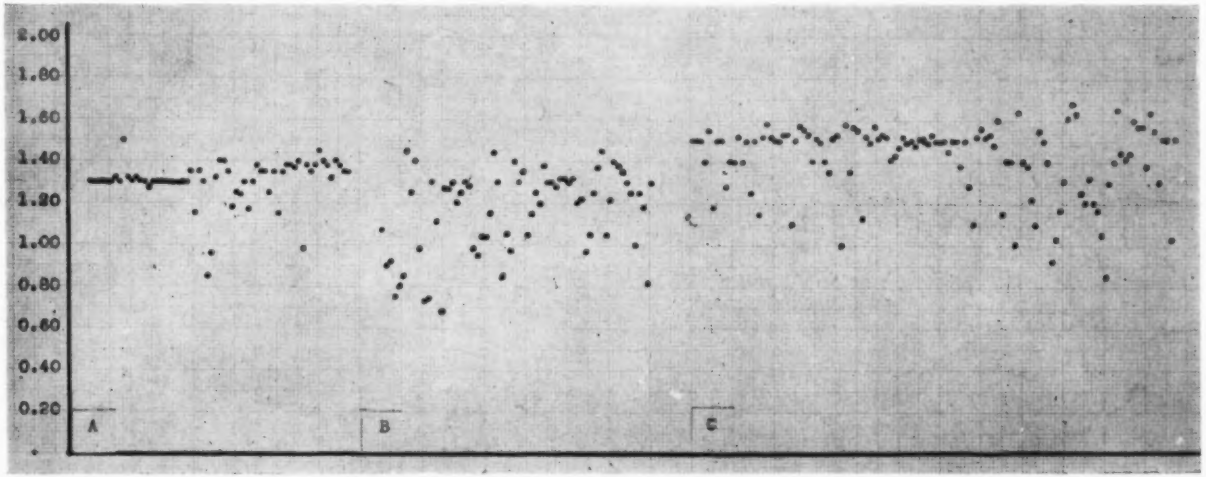

Fig. 7.—Chart of a man of 67 years with coronary sclerosis and acute auricular fibrillation treated with folium digitalis and quinidine. Sinus rhythm returned the following day, revealing a second degree A-V block with dropped ventricular beats (4:3 and 5:4 A-V block). *A*, was during rest, *B* after exercise, *C*, during rest. Note the distinct "ceiling" level after *A* and *C*. Scale to the left: 100ths of a second. (Patient A. B.)

all the cases, and it was not much influenced by the ventricular rate. Their presence usually became more obvious during periods of changing ventricular rates and the apparently smooth transitions between high and low rates could then be seen to take place between these two to four fixed levels.

It may seem surprising that this system had not been observed earlier. This is probably due to the fact that the detection of the phenomenon requires the use of a method which makes it possible to observe the changes in the R-R intervals during periods of changing ventricular rates.

An explanation for one type of such levels has already been suggested (Fig. 7). An upper limit for the length of R-R intervals in absolute arrhythmia is set by the intrinsic rate of the atrioventricular node and the ventricles. This manifests itself as escaped beats, and R-R intervals of this type may form a distinct level at 1.20 to 2.00 seconds as a "ceiling" or upper limit for the distribution of the dots.

The corresponding "floor" or lower limit (Level I) was the level most constantly present. It was distinctly visible in most cases with a ventricular rate above 100 per minute. At lower rates the dots of this area were often too few to form a distinct level. In high-grade tachycardia it was usually found at 0.35 second, but with decreasing rates it rose usually to about 0.40 second, where it remained (Patient D. M. being the sole exception). This level could be observed in twenty of the thirty-four cases.

Tachycardias in which the intervals are limited to Level I correspond well to the theory of Hoffmann. There is little reason to doubt that the ventricular beats which form Level I have been elicited by the first fibrillation impulse passing through the atrioventricular node after the end of its refractory period.

The rate of auricular fibrillation is usually stated to be 300 to 500 per minute, corresponding to a fibrillation period (F-F interval equals interval between the fibrillation impulses) of 0.12 to 0.20 second. Judging from these figures the intervals of Level I may correspond to two fibrillation periods. Consequently the duration of the nodal refractory period should be between one and two fibrillation periods in duration.

Accordingly, Level I may be regarded as the level of a 2:1 atrioventricular block in auricular fibrillation. It is tempting to interpret analogously Level II as corresponding to a 3:1 and Level III to a 4:1 atrioventricular block. However, this hypothesis seems unlikely from a study of Fig. 8.

This chart may be regarded as an ideal realization of the above theory. It is derived from a case of auricular flutter that apparently shows absolute ventricular arrhythmia. As compared with cases of auricular fibrillation, such a case offers the advantage that the auricular period (= flutter period = F-F interval) is of constant duration and may be measured accurately. The R-R intervals of this chart form very distinct levels, and it is evident that the step from zero to the first level (probably comparable with Level I of the fibrillation cases) corresponds to two flutter periods and that the steps between the following levels are each equal to one flutter period. These levels may thus be regarded as true expressions of 2:1, 3:1, 4:1, and 5:1 block.

Fig. 8.—Chart of a man of 57 years with hypertensive heart disease and auricular flutter. *A* to *B* was before exercise; *B* to *C*, moderate exercise; after *C*, post-exercise tachycardia. F-F interval 0.22 second (scale to the right). Scale to the left: 100ths of a second. (Patient B. H.)

At first glance Fig. 3 (patient A. W.) may seem essentially similar to the illustration of the flutter case. There are, however, two important differences. The first one is that the steps I to II and II to III are considerably higher in the former (0.32 as against 0.22 second). The reverse was to be expected, fibrillation being essentially an activity of higher frequency than flutter. The second difference is that steps I to II and II to III in the fibrillation case are considerably greater than half step O to I. If step O to I corresponds to two fibrillation periods, then steps I to II and II to III cannot correspond to one fibrillation period.

These findings are by no means peculiar to patient A. W. In all fibrillation cases the steps between Levels I, II, and III were greater than 0.22 second (usually between 0.25 and 0.30 second). Fibrillation periods of this duration should correspond to fibrillation rates of 240 to 200 per minute. In three of these cases the F waves were at times sufficiently distinct to permit measurement of the F-F intervals. The mean values of twenty-five intervals in each of these cases were 0.16 ± 0.03, 0.17 ± 0.03, and 0.18 ± 0.03 second. These figures correspond to fibrillation rates of 370 to 330 per minute, values within the normal range. There is thus considerable evidence against the assumption that the fibrillation period is the primary unit in this scale of levels.

The author suggests as a working hypothesis that the unit sought for is the refractory period of the atrioventricular node. It was concluded earlier in this paper that the refractory period of the atrioventricular node is probably longer than one but shorter than two fibrillation periods. Its duration is thus probably of the order 0.25 to 0.35 second, and this would be compatible with the above hypothesis. Some further comment is necessary, however, to show how the hypothesis works.

It is quite possible that under certain conditions the atrial impulse may enter the atrioventricular node and pass some distance down the atrioventricular system but fail to reach the ventricular myocardium. Such conditions were shown by Lewis and Master[3] to be present for some time ("phase of interference") after the end of the absolute refractory period of the atrioventricular node. If the fibrillation impulse reaches the atrioventricular node within this period, the result will be that the ventricular systole fails to appear and that the ventricles must wait for a new activating impulse until the end of another nodal refractory period.

R-R intervals formed in this way will be approximately equal to two nodal refractory periods. If they occur often (i.e., if the phase of interference is long) they will form a level about one nodal refractory period above Level I. After the end of the second refractory period the next fibrillation impulse has again the same chance of arriving at the node within the phase of interference. The result will be R-R intervals made up of approximately three nodal refractory periods, and a corresponding level in the charts (Level III). In this way a system of levels may be formed in which the refractory period of the atrioventricular node is the primary unit.

If this is the manner in which Levels II and III are formed, then steps I to II and II to III should be equal to the step O to I rather than to the nodal refractory period itself (the duration of which cannot be exactly measured). The

charts show that the steps I to II and II to III are actually slightly smaller than the step O to I. However, it must be stressed that most complicating factors (differences of atrioventricular conduction time, etc.) tend to prolong short R-R intervals and shorten long ones (cf. similar experiences in the study of sino-auricular block).

The author at present regards this theory as the most plausible one. Although not entirely satisfactory, it has the advantage that it offers the best explanation for the presence of very long R-R intervals in absolute arrhythmia.

SUMMARY

In cases of auricular fibrillation with absolute ventricular arrhythmia a great number of R-R intervals were measured. By plotting the intervals in special charts the author was able to show that in many cases the intervals had a tendency to concentrate at certain levels. Several such levels were often present in the same case, and the position of the levels was strikingly constant from case to case. The position of these levels was also remarkably independent of the ventricular rate. Usually the levels were clearly distinguishable during periods of changing ventricular rates, and apparently smooth transitions between high and low rates could be seen to take place between two to four fixed levels.

The formation of these levels is probably not to be explained by a single mechanism. The importance of several factors, fibrillation period, nodal refractory period, and ventricular (nodal) intrinsic automatism, is discussed.

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RETROGRADE CONDUCTION TO THE AURICLES IN PAROXYSMAL VENTRICULAR TACHYCARDIA

A Report of an Unusual Case Diagnosed by the Esophageal Lead

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VENTRICULAR tachycardia is a subject which has appeared in the literature with increasing frequency during the past decade. In most recent reports the activity of the auricles is of secondary importance and is often not discussed. Rarer still is there mention of retrograde auricular conduction during ventricular tachycardia. It is the purpose of this paper to present a case of probable ventricular tachycardia in which retrograde ventricular conduction to the auricles was seen. We say probable ventricular tachycardia because there seems to be at the present time no sure method of differentiating ventricular tachycardia without retrograde block from auricular tachycardia with ventricular aberration. We have also reviewed from the literature eighty-one cases of ventricular tachycardia with our attention directed toward the rate of the auricles in order to obtain some indication of the frequency of retrograde conduction. Above all, we wish to emphasize the importance of the esophageal lead in the diagnosis of obscure tachycardias.

CASE REPORT

A 64-year-old white man entered Providence Hospital on Nov. 2, 1949, complaining of intermittent pain in his lower abdomen for the preceding two days. The pain was not severe and did not radiate to his chest or down his arms. He noticed that his heart rate was unusually rapid on the day before admission and he experienced orthopnea and exertional dyspnea for the first time in his life on the day of admission.

The past history is significant in that he had had two attacks of coronary thrombosis four years before, from which he had made uneventful recoveries. There were no symptoms of cardiac failure until the day of the present admission, and he had never taken digitalis. There was no history of hypertension or angina. At the age of 17 years he had been ill with what was considered to be rheumatic fever.

Physical examination revealed a well-developed, pale man in no acute distress but who appeared acutely ill. The temperature was 97°F. The pulse was 120 and regular, and the blood pressure was 90/70 mm. Hg in both arms. He was having mild respiratory difficulty and was unable to lie flat in bed. The lungs were normal to percussion. Moist inspiratory râles were heard at the right base. The heart was moderately enlarged to percussion. The heart sounds did not vary in intensity. No murmurs were heard. The liver edge could be felt on deep inspiration. There was no ankle edema. The diagnosis at this time was acute myocardial infarction.

Laboratory examination showed a hemoglobin of 11.9 Gm. or 82 per cent, R.B.C. 3,840,000, W.B.C. 7,100 with a normal differential count. Urinalysis revealed no abnormalities. The Mazzini test was negative, and the sedimentation rate was 45 mm. at the end of one hour. A

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chest x-ray showed a moderately enlarged heart and clear lung fields. An electrocardiogram (see Fig. 1) showed this interesting tracing. No definite diagnosis could be made from this record, but the impression was that this could represent either an auricular tachycardia or flutter with a bundle branch block, a nodal tachycardia with a bundle branch block, or ventricular tachycardia.

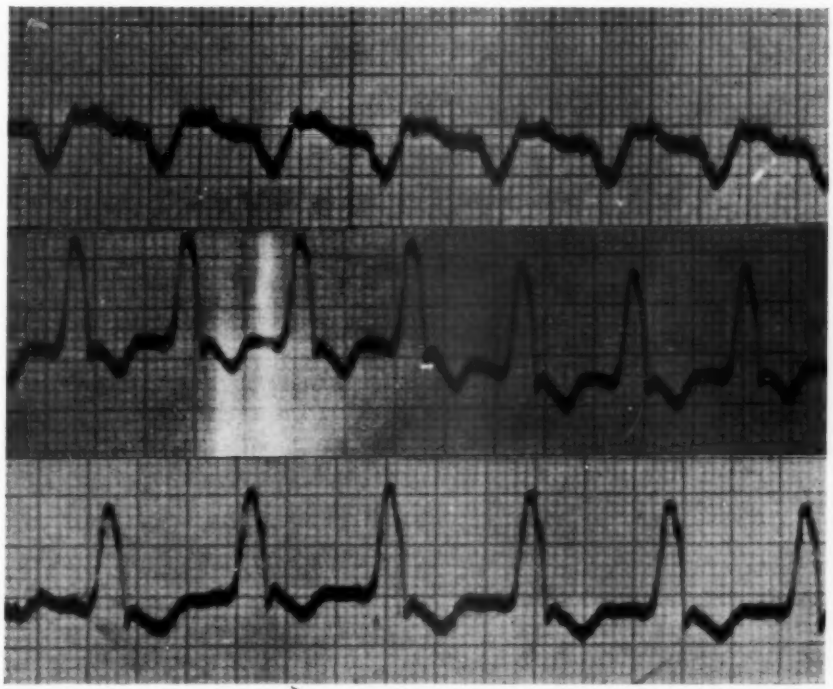

Fig. 1.—Leads I, II, and III. Rate 125. A bizarre tachycardia is present. P waves are not identified.

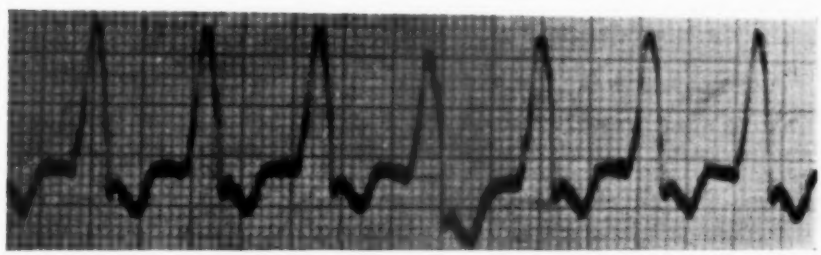

Fig. 2.—Lead II. Note a widening of the QRS complex after quinidine therapy.

The patient was placed on strict bed rest, salt-free diet, mercurials, and oral papaverine. He was given 1.0 Gm. of quinidine in divided doses daily in an effort to stop the tachycardia. Under this regime the patient improved subjectively. However, his tachycardia persisted. An electrocardiogram taken on the third hospital day (see Fig. 2) showed what appeared to be an increase in the QRS interval which we felt was the result of the quinidine. Therefore, the quinidine was discontinued and the patient was placed on full doses of digitoxin. During this period

ocular and carotid pressure, as well as intramuscular injections of Mecholyl, were employed unsuccessfully in attempting to halt the tachycardia. The blood pressure remained at 90/70 mm. Hg during this period.

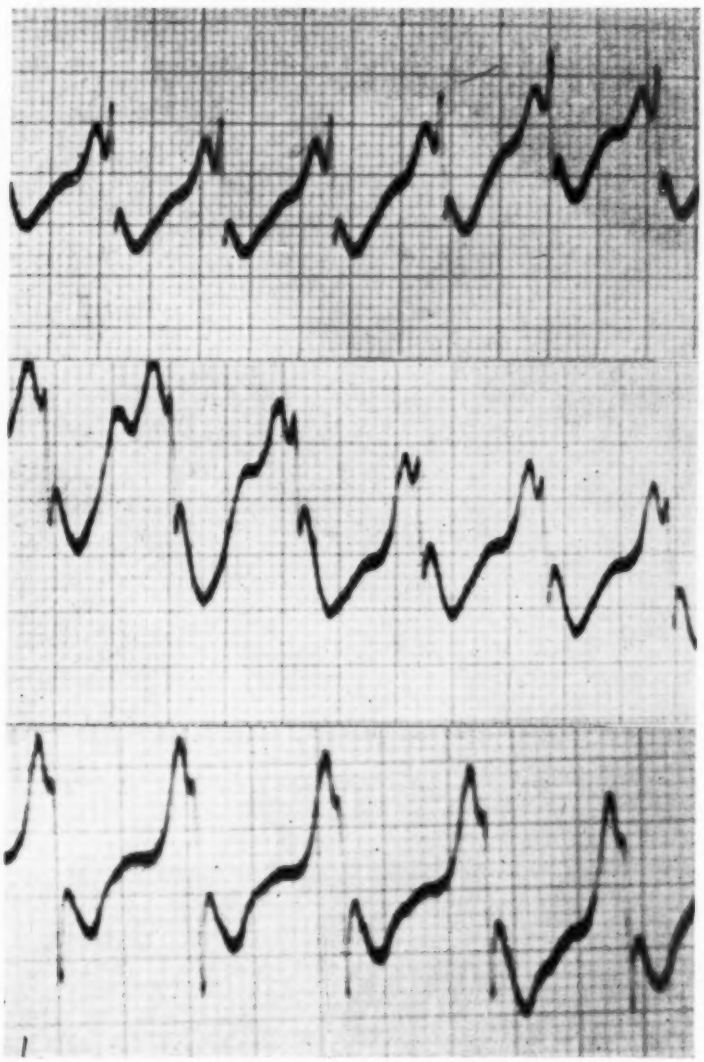

Fig. 3.—Esophageal Leads *A*, *B*, and *C*. In *A* the esophageal electrode is at the level of the auricle, and a tall, spiked P wave emerges from the descending limb of the ventricular complex. In *B* the electrode has descended further down the esophagus, and the P wave becomes less prominent. In *C* the electrode is at the level of the ventricle, and the P wave has fused with the ventricular complex.

On the ninth hospital day an electrocardiogram using the esophageal lead was performed in order that the auricular activity might be better ascertained. The unusual record shown in Fig. 3 was seen. The standard leads are similar to those previously taken. When the esophageal electrode is at the level of the auricles, the P wave, which is hidden in the standard leads, is seen to emerge from the descending limbs of the preceding ventricular complex. As the electrode descends down to the ventricular level, the ventricular complex becomes larger and the P wave becomes

smaller and is eventually hidden by the ventricular complex. This tracing suggests the diagnosis of ventricular tachycardia with retrograde P waves or possibly nodal or auricular tachycardia with ventricular aberration.

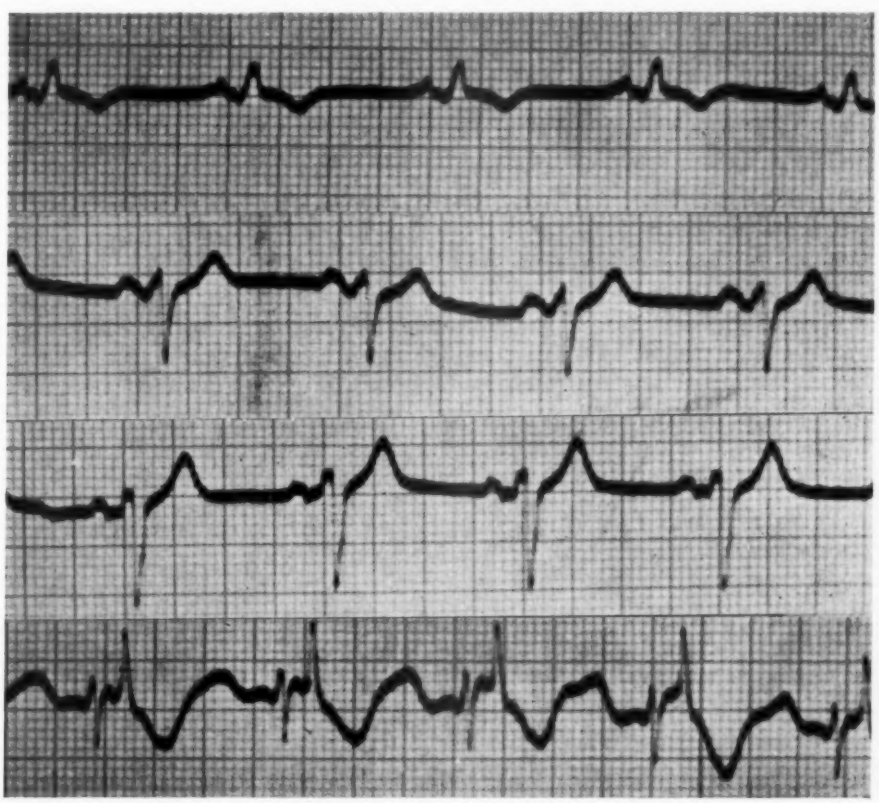

Fig. 4.—Leads I, II, III, and esophageal lead. Taken shortly after reversion to normal rhythm. Rate is 75, P-R interval is 0.14 second, QRS interval is 0.09 second. Note that in the esophageal lead each QRS complex is now preceded by a smaller, diphasic P wave, demonstrating the normal auriculo-ventricular relationship in the esophageal lead.

During the next six days the patient received 0.3 mg. of Digifortis daily which was stopped on the seventeenth hospital day because of nausea. On the next day the rhythm returned to normal. His blood pressure was 115/80 mm. Hg, and he made a rapid recovery thereafter. The electrocardiogram showed the following record (see Fig. 4). Note that the QRS is of normal duration and totally unlike that seen during the tachycardia. The esophageal lead demonstrates the normal relationship of auricular and ventricular complexes. At no time during his illness did he have chest pain or fever. He was discharged on the twenty-first hospital day.

DISCUSSION

We wish to emphasize the use of the esophageal lead in the diagnosis of obscure tachycardia. It depicts the auricular wave more accurately than does any other lead. When the electrode is at the level of the auricle, the P wave assumes an entirely new appearance. It is broken up into two components.

The first is a sharp, diphasic wave which has an initial upward deflection. It closely resembles the QRS of the ventricle, and probably represents the spread of the excitation wave through the auricle. The initial deflection time has been below 0.04 second in our records, and is usually about 0.03 second. This wave is characteristic and is easily recognized. The second component would correspond to the ventricular T wave and is called the T_a wave. This wave is not easily seen in most tracings, especially with the tachycardias. The auricular waves in our case were quite typical. Therefore the direction of activation would be toward the recording electrode. Although we believe the auricular waves to be retrograde from the ventricle, they were no different in configuration from those originating in the S-A node seen in other cases.

In the esophageal electrocardiogram (Fig. 3) the auricular complex is seen to emerge directly after the peak of the ventricular QRS. A considerable period of time (0.28 second) separates each auricular complex from what we interpret as the beginning of the following ventricular QRS. We feel that this would favor the diagnosis of retrograde auricular conduction. Further evidence against this being auricular tachycardia is that the patient did not respond to eyeball or carotid pressure. Intramuscular Mecholyl had no effect on his heart rate. These measures will usually, but not invariably, arrest a case of auricular tachycardia.

SUMMARY OF THE CASES REVIEWED

The Auricular Rhythm in Ventricular Tachycardia.—In half of the cases reviewed, no auricular waves could be identified. This differs somewhat from the results obtained in a similar analysis by Prinzmetal and Kellogg,[32] who found that only ten of the sixty-eight cases which they reviewed showed absent P waves. In explaining this apparent discrepancy, we would point out that most of the cases which we reviewed are relatively recent. During the past decade the tendency has been to use less rigid diagnostic criteria and to pay less attention to the auricular rate in ventricular tachycardia. Again we emphasize the value of the esophageal lead in demonstrating hidden auricular waves. Otherwise our results would correspond to their work. Another explanation for the low incidence of auricular activity noted is that an unknown number of these patients might have had auricular fibrillation unbeknown to the physician before the tachycardia developed.

TABLE I

Total number of cases reviewed	81
Auricular waves not seen	40
Auricular fibrillation (noted previously)	14
Independent auricular rhythm	18
Retrograde heart block (2:1 etc.)	6
Retrograde conduction	3

The Effects of the Rate of the Ventricles Upon Auricular Activity.—Sixty of the cases reviewed had a ventricular rate of over 160, and twenty-one had a

ventricular rate of below 160. Independent auricular beats were seen in both the rapid and the slow types of ventricular tachycardia with about equal frequency. All the cases of retrograde heart block occurred when the ventricular rate was over 160. All the cases of retrograde auricular conduction (including our own) occurred when the ventricular rate was below 160. This would indicate that retrograde heart block can be, in part at least, a function of the ventricular rate. The auricle simply cannot respond to each ventricular beat when the ventricular rate is extremely rapid.

TABLE II.*

	NO HEART DISEASE	DIGITALIS INTOXICATION	HEART DISEASE
Auricular waves not seen	5	5	28
Auricular fibrillation	0	3	11
Independent auricular rhythm	1	3	13
Retrograde heart block	2	2	2
Retrograde conduction	3	0	0
Total	11	13	54

*From seventy-eight cases.

The Effect of Heart Disease Upon Auricular Activity.—Only eleven of the seventy-eight cases reviewed showed no previous evidence of heart disease. One of these cases showed an independent auricular rhythm. The ventricular rate in this case was 260. Two cases in this group demonstrated retrograde heart block. Likewise, they were associated with rapid ventricular rates, 170 and 272. Of possible significance is that all three cases of retrograde conduction occurred in the group which showed no heart disease. Our case would appear to be an exception, still, our patient had never been in failure nor received digitalis, and the etiology of his tachycardia was not determined.

DISCUSSION

Sir Thomas Lewis[5] produced ventricular tachycardia experimentally by the ligation of the coronary arteries in dogs. The tachycardia was usually heralded by ventricular extrasystoles occurring first as single beats, then in pairs, and finally in succession. Generally, after a few successive extrasystoles retrogression to the auricles took place. The retrograde conduction seemed to be largely independent of the rate of the sinus rhythm but occurred somewhat more frequently when the rate was slow. Once retrograde impulses made their appearance, they occurred with increasing frequency. From his records it seems that retrograde impulses were usually, but not invariably, present. He noted that whenever the ventricular rate exceeded 220, the auricles, which formerly had responded to every ventricular beat, now responded to every second or third beat.

There are several factors which we feel are probably responsible for the low incidence of retrograde conduction seen clinically. Scott[7] has shown that retrograde conduction can occur only when the auricle is responsive. If the

auricle has already contracted from the normal sinus impulse when the retrograde impulse appears from the ventricle, no retrograde contraction will take place. Undoubtedly sinus interference, especially when the rate is rapid, will inhibit retrograde conduction, but this mechanism alone does not explain the low incidence.

There is both clinical and experimental evidence that a rapid ventricular rate of itself will produce a retrograde heart block.

Finally, the large majority of patients with ventricular tachycardia are found to have arteriosclerotic heart disease or digitalis intoxication. This may be quite significant, since both of these conditions are commonly associated with impaired conduction.

CONCLUSIONS

1. A case of probable ventricular tachycardia with retrograde auricular conduction is reported. The importance of the esophageal lead in the diagnosis of obscure tachycardia is stressed.

2. A series of eighty-one cases of ventricular tachycardia was analyzed for the auricular rate. In half the cases no auricular waves were seen. Eighteen showed an independent auricular rhythm, fourteen showed auricular fibrillation. Retrograde auricular conduction was seen in nine cases, six of which had a retrograde partial heart block. In only three cases was pure retrograde conduction observed.

3. The experimental production and clinical incidence of retrograde auricular conduction have been discussed.

We are indebted to Mrs. Patricia Roundtree for her technical assistance in preparing this paper.

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A STUDY OF THE V LEADS IN CONGENITAL HEART DISEASE

With Particular Reference to Ventricular Hypertrophy and Its Diagnostic Value

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The development of surgical techniques for the correction of certain congenital cardiac defects has provided a great stimulus for better diagnosis of such conditions, and the literature on this subject has grown accordingly. Numerous studies have appeared, particularly with reference to the use of venous catheterization of the heart and Diodrast angiocardiograms as diagnostic aids. Surprisingly little, however, has been written regarding the various electrocardiographic changes that one may expect to find in congenital heart disease. Older studies based on the standard leads only[1] provided valuable information but were not directed primarily toward establishing the role of the electrocardiogram in deciding whether or not surgery might be indicated in these cases. Very little information is available regarding the value of the unipolar limb and precordial leads in the diagnosis of congenital heart disease.[2,3] Therefore, a study was undertaken of a series of patients with congenital heart disease, all of whom had had unipolar extremity and precordial electrocardiograms.

Table I. A Summary of 153 Cases With Proved Diagnosis of Congenital Heart Disease and Complete Unipolar Electrocardiograms

Acyanotic		82
Patent ductus arteriosus, uncomplicated	39	
Coarctation of the aorta	32	
Interauricular septal defect	8	
Interventricular septal defect	2	
Subaortic stenosis	1	
Cyanotic		71
Tetralogy of Fallot	40	
Tetralogy plus additional lesions	5	
Transposition of great vessels	5	
Trilocular heart	3	
Eisenmenger's complex plus additional lesions	3	
Tricuspid atresia with rudimentary right ventricle	4	
Truncus arteriosus	4	
Miscellaneous	12	

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SUBJECTS AND METHOD

In order to assure reasonably accurate diagnosis, patients were included only when they had had exploratory thoracotomy or post-mortem examination or when they provided adequate clinical and x-ray evidence for definite diagnosis.

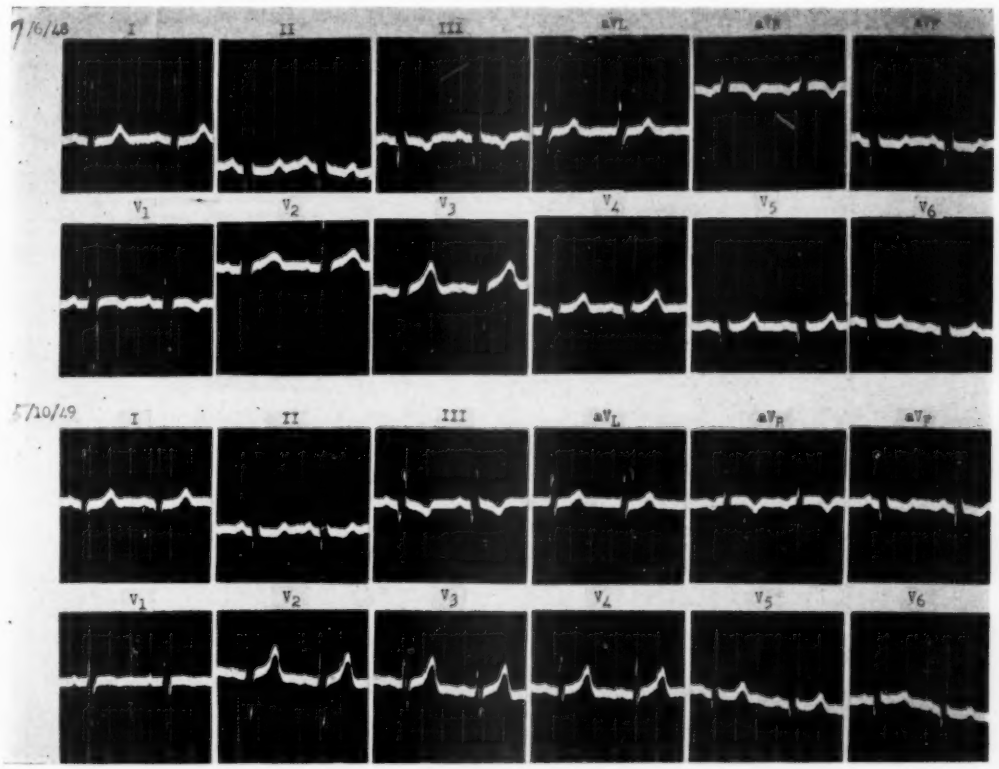

Fig. 1.—A. B. C., a girl aged 15 years, had patent ductus arteriosus. Ductus with lumen of 1.5 cm. was ligated July 21, 1948. Note changes in voltage of the QRS complexes between record of July 6, 1948, and May 10, 1949. Cardiac enlargement was plus 14 per cent before surgery.

There were 195 such patients studied at the University of California Hospital from January, 1946, to January, 1950. Of these, 153 had had preoperative and/or ante-mortem unipolar electrocardiograms (Table I). Thirty-eight of the 153 patients came to post-mortem examination. Electrocardiograms were reviewed in detail according to standards published elsewhere.[3,4]

RESULTS

1. *Acyanotic Patients.*—

A. *Uncomplicated patent ductus arteriosus:* Complete V-lead examination was made on thirty-nine patients in whom the diagnosis was later verified by operation (thirty-eight patients) or at post-mortem examination (two patients) (Figs. 1 and 2). The electrocardiogram was normal in twenty-one. There was

evidence of left ventricular hypertrophy in seventeen. In one case, delayed ventricular activation in V_1 suggested right bundle branch block.

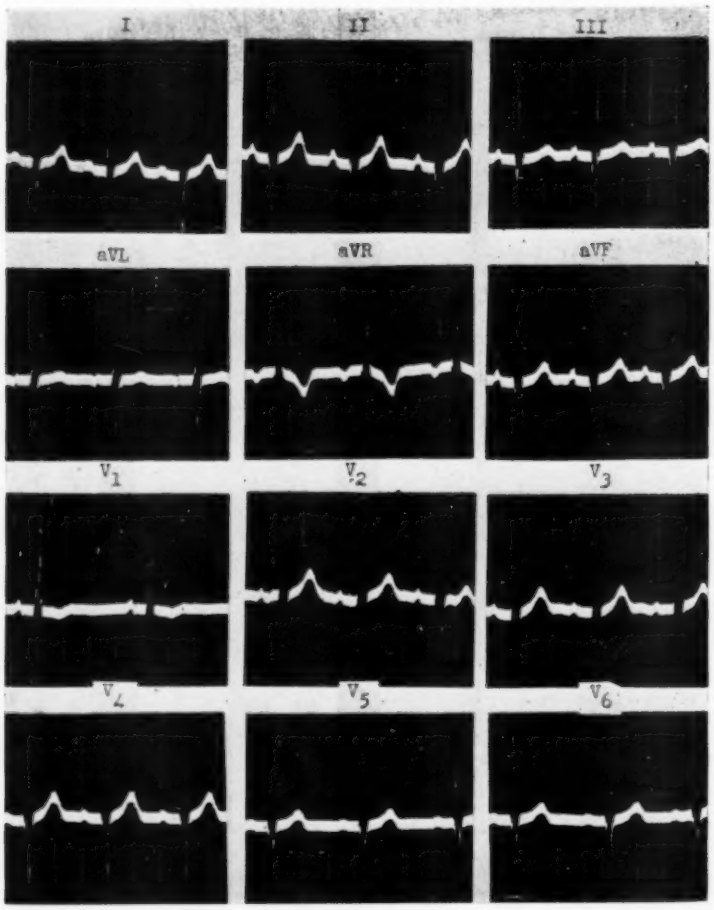

Fig. 2.—A. D. W., a boy aged 5½ years, had congenital heart disease thought clinically to be a patent ductus arteriosus. At exploratory thoracotomy an intracardiac thrill at the base of the pulmonary artery was found. Right ventricular hypertrophy in the electrocardiogram was evidence against a patent ductus arteriosus.

An attempt was made to correlate the electrocardiographic findings with the size of the patent ductus. In the cases where the electrocardiogram was normal, the ductus was found to be less than 1 cm. in diameter. In twelve cases where the ductus was found to be larger than 1 cm., evidence of left ventricular hypertrophy was found in nine. It was therefore concluded that left ventricular hypertrophy in patent ductus arteriosus is a function of the width of the ductus, and hence a guide to the size of the shunt.

In one patient the diagnosis of patent ductus was suggested, but the patient had electrocardiographic evidence of right ventricular hypertrophy and retro-

grade arteriograms failed to show the lesion. At operation no ductus was found but there was a thrill in the pulmonary artery and the pressure here was 32 cm. of water. This patient probably had an interauricular septal defect.

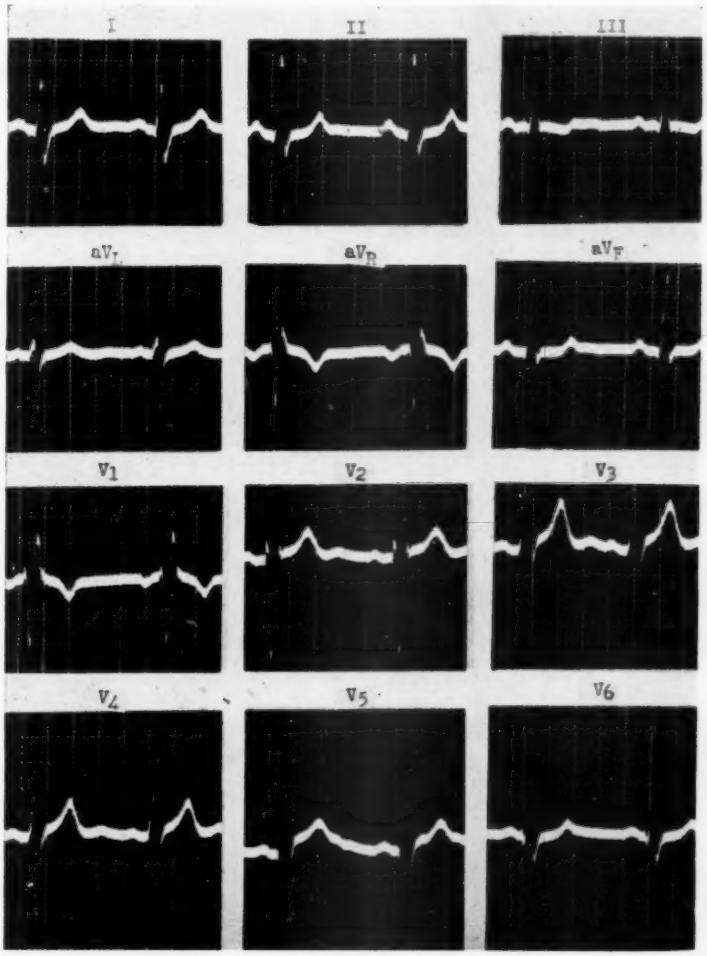

Fig. 3*A*.—W. M. R., a boy aged 24 years, had coarctation of the aorta. Aortectomy was performed on Oct. 23, 1948. High-voltage QRS complexes suggest left ventricular hypertrophy. QRS of 0.13 second with late R^1 in V_1 suggests incomplete right bundle branch block. Cardiac enlargement was plus 40 per cent.

B. *Coarctation of the aorta:* In thirty-two patients, coarctation of the aorta was diagnosed either by post-mortem examination (nine cases), at operation (nine cases), by retrograde carotid arteriography (six cases), or by unequivocal clinical findings (eight cases). The electrocardiogram was normal in six. It showed evidence of left ventricular hypertrophy in nineteen and right bundle branch block with left ventricular hypertrophy in two (Fig. 3,*A*). Five patients showed evidence of right ventricular hypertrophy, but all of these had some

additional defect (interauricular septal defect in two, interventricular septal defect in one, congenital mitral stenosis in one, and patent foramen ovale in one). (One such example is seen in Fig. 3,*B*.)

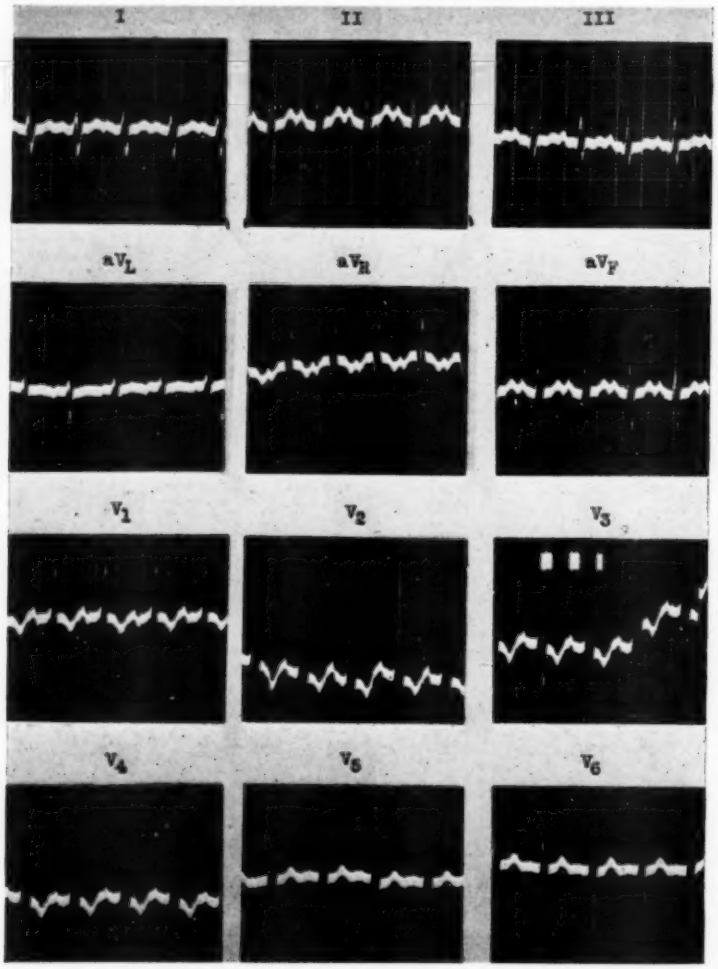

Fig. 3*B*.—R. B. M., a male infant, aged 13 months, had coarctation of the aorta with cardiac failure and right ventricular hypertrophy. Autopsy confirmed right ventricular hypertrophy (right ventricle, 8 mm.). There was a large patent foramen ovale (0.5 cm.).

C. *Interauricular septal defect:* There were eight cases of proved interauricular septal defect, one by autopsy, one at operation, four by cardiac catheterization, and two by Diodrast angiocardiograms. Five patients had electrocardiographic signs of right ventricular hypertrophy and three had incomplete right bundle branch block. No patient had left ventricular hypertrophy. There were two patients with interventricular septal defect, one had a normal

record, and one showed left ventricular hypertrophy. Many other patients who probably had septal defects were not included in this paper because the diagnosis was made solely on the basis of physical signs.

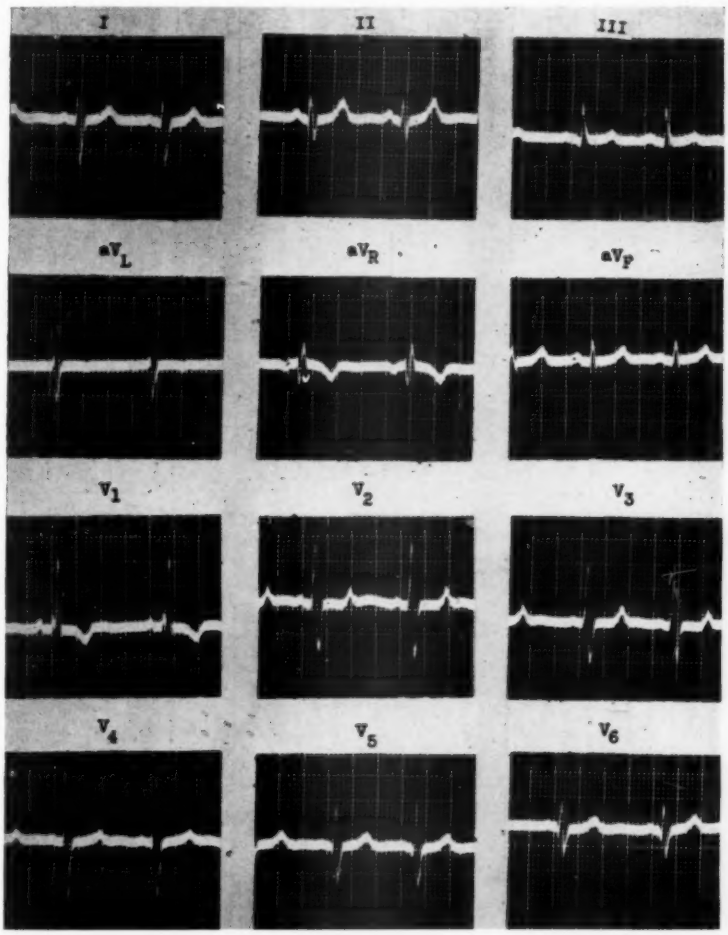

Fig. 4.—F. L., a boy aged 7 years, had congenital heart disease with pulmonary atresia. Marked right ventricular hypertrophy at autopsy.

2. *Cyanotic Patients.*—

A. *Tetralogy of Fallot:* The electrocardiogram revealed right ventricular hypertrophy in thirty-nine of the forty patients with tetralogy of Fallot. The one exception was a 3½-month-old boy (T. O.) whose electrocardiogram suggested right bundle branch block more than right ventricular hypertrophy. The axis was 100 degrees. The boy died 2 years later, and the diagnosis of tetralogy of Fallot was confirmed at autopsy.

TABLE II. SUMMARY OF AUTOPSIED CASES OF CONGENITAL HEART DISEASE

NO.	NAME	AGE	SEX	ELECTROCARDIOGRAM	OPERATION	AUTOPSY FINDINGS
1.	C. S.	34 yr.	M	LVH and anterior septal myocardial infarct.	Aortectomy, lumen 25 mm.; died 11 days postoperatively.	Postoperative coarctation; weight 500 Gm.; RV, 9 mm.; LV, 19 mm; coronary sclerosis; patchy fibrosis of septum, aortic stenosis.
2.	W. S.	16 yr.	M	LVH.	Subclavian to aorta, lumen 3 to 4 mm.; died 28 days postoperatively.	Postoperative coarctation; weight, 300 Gm.; RV, 6 mm.; LV, 20 mm.; no myofibrosis; coronary arteries within normal limits.
3.	R. M.	1 yr.	M	RVH.	Aortectomy, lumen 1 mm.; died coming out of anesthesia.	Postoperative coarctation; atelectasis left lung; anastomosis intact; patent foramen ovale 0.5 cm.; weight 120 Gm.; RV, 8 mm.; LV, 10 mm.
4.	J. L.	7 yr.	M	RVH.	Exploratory; died 2 hr. postoperatively.	RVH and LVH; RV, 12 mm.; LV, 12 mm.; overriding of aorta; pulmonary atresia; anomalous right vessel; PDA; IV septal defect.
5.	M. D.	3 yr.	F	Suggestive of RVH.	Blalock; died of acute leucemia 2 yr. later.	Tetralogy; acute leucemia; RV, 10 mm.; LV, 12 mm.
6.	R. B.	2 yr.	F	RVH.	Died pulmonary edema after Diodrast.	Tetralogy; weight 45 Gm.; RV, 11 mm; LV, 7 mm.
7.	D. S.	3 mo.	F	RVH.	Exploratory; died at operation.	Transposition of great vessels; two IV septal defects; PDA; RV, 5 mm.; LV, 6 mm.
8.	J. C.	23 yr.	M	RVH.	Incomplete Blalock; died at operation.	Tetralogy; RV, 18 mm; LV, 19 mm.
9.	J. D.	8 yr.	F	RVH.	Blalock; died 2 days postoperatively.	Transposition of great vessels; pulmonary stenosis; IA septal defect; RV, 9 mm; LV, 7 mm.
10.	M. F.	24 yr.	F	RVH.	Blalock; died 5 hr. postoperatively.	Tetralogy; rheumatic tricuspid valve; RV, 15 mm.; LV, 10 mm.
11.	J. C.	21 mo.	M	RVH.	Exploratory; died 20 days postoperatively; atelectasis.	Truncus arteriosus; IV septal defect; RVH; RV, 5 mm.; LV, 6 mm.
12.	G. B.	2 yr.	F	RVH.	Exploratory; died suddenly 1½ hr. postoperatively.	Transposition of great vessels; single ventricle, 7 mm.; Pulmonary stenosis; IA septal defect; left pulmonary vein to right auricle.

13.	L. T.	21 mo.	M	RVH.	Pott's operation; died 1 hr. postoperatively.	Tetralogy with right arch; RV, 10 mm.; LV, 10 mm.
14.	V. M.	3 yr.	M	RVH.	Coarctation with no lumen; died 2 mo. later; cardiac failure.	Postoperative coarctation; possible congenital mitral stenosis; slight aortic stenosos; anastomosis intact; RV, 7 mm.; LV, 18 mm.
15.	G. K.	19 yr.	M	Abnormal record with right axis deviation; not typical of hypertrophy of either ventrical.	Blalock; died at operation while pulmonary artery clamped.	Marked overriding of aorta; large IV septal defect; small right pulmonary artery from RV; ductus from aorta to left lung; two superior venal cavae to right auricle; large aneurism, ductus at left hilus; RV, 11 mm.; LV, 13 mm.
16.	F. S.	3½ mo.	F	Dextrocardia with RVH.	None.	Died progressive dyspnea and cyanosis (elsewhere); complete dextroposition all organs; nearly complete absence IV septum; RVH; no measurements.
17.	T. O.	2½ yr.	M	RBBB plus? RVH (2 yr. before death).	None.	Tetralogy; died thrombosis left inferior carotid with cerebral infarct; no measurements.
18.	B. H.	17 weeks	F	RVH.	None.	IV septal defect (1 cm.); PDA; coarctation; multiple congenital defects; RV, 7 mm.; LV, 5 mm.
19.	P. S.	2 weeks	F	Probably within normal limits.	None.	Died with respiratory failure; coarctation; large PDA; large globular heart enlarged to right and left; RV, 4 mm.; LV, 5 mm.
20.	W. S.	8 mo.	M	RVH.	None.	Small aorta; absent isthmus; no communication from arch to descending aorta; IV septal defect 7 mm.; large pulmonary artery; RV, 10 mm.; LV, 9 mm.; PDA, 8 mm. to descending aorta.
21.	R. S.	7½ mo.	M	RVH.	None.	Patent foramen ovale; IV septal defect; PDA; large pulmonary artery; RV, 6 mm.; LV, 5 mm.
22.	C. G.	4 yr.	F	RVH.	None.	Single auricle; IV septal defect; pulmonary stenosis; RV, 10 mm; LV, 10 mm.
23.	I. M.	18 mo.	F	RVH.	None.	Tetralogy; bicuspid pulmonary valve; RV, 9 mm.
24.	M. G.	5½ yr.	M	Probable LVH; not typical.	None.	Patent ductus; LVH; heart weight 110 Gm. (normal, 73 Gm.); died atresia bile duct; progressive cirrhosis; RV, 3 mm.; LV, 16 mm.
25.	R. G.	35 yr.	M	Probable LVH.	Aortectomy.	Postoperative coarctation; anomalous coronary artery; bicuspid aortic valve; LVH; RV, 5 mm.; LV, 25 mm.

(Table continued on next page.)

TABLE II. SUMMARY OF AUTOPSIED CASES OF CONGENITAL HEART DISEASE—CONT'D

NO.	NAME	AGE	SEX	ELECTROCARDIOGRAM	OPERATION	AUTOPSY FINDINGS
26.	L. C.	27 yr.	F	RVH.	None.	Pulmonary stenosis with dilated pulmonary artery; IA defect 1 cm. wide; hypoplastic aorta; RV, 15 mm.; LV, 11 mm.
27.	L. H.	28 mo.	F	RVH.	None.	Tetralogy; patent foramen ovale; bicuspid pulmonary valve, RV, 6 mm.; LV, 5 mm.
28.	K. W.	2 yr.	F	LVH.	Blalock.	Large IA septal defect; small pulmonary artery; heart weight 81 Gm.; IV, septal defect; RV small, 5 c.c. vol., 3 cm.; LV large.
29.	K. B.	3 mo.	F	RVH.	None	Transposition of great vessels; IV septal defect 1 cm.; slight overriding aorta; RV, 7 mm.; LV, 7 mm.
30.	J. S.	15 mo.	F	RVH.	None.	Tetralogy and patent foramen ovale 2 mm.; IV septal defect 1.3 by 0.5 cm.; RV, 5 mm.; LV, 8 mm.
31.	D. S.	1 yr.	F	LVH.		Coarctation of aorta with LVH (Coroner).
32.	L. P.	21 days	M	LVH.		Transposition great vessels; weight 60 Gm.; absent tricuspid valve; PDA, 1 cm. diameter; coarctation of aorta; IV septal defect; hypoplastic right ventricle; LV, 9 mm., dilated and hypertrophied.
33.	J. W.	1 mo.	F	RVH; possible associated LVH.		Infantile coarctation 5 mm.; PDA, 8 mm.; IA, septal defect 1 cm. in diameter; RV, 5 mm.; LV, 10 mm.
34.	R. H.	4 mo.	M	RVH.		Infantile coarctation; PDA, 5 mm.; weight, 67 Gm.; IV septal defect 0.5 cm.; RV, 5 mm. dilated; LV, 7 mm.
35.	C. J.	8 weeks	F	RVH.	Aortectomy.	Coarctation of aorta; weight 60 Gm.; IV septal defect 6 mm. diameter; RV, 8 mm.; LV, 8 mm.
36.	L. C.	2½ yr.	M	RVH.	Extracardiac anastamosis of greater and lesser circulation.	Transposition of great vessels; weight, 105 Gm.; high IV septal defect 1 cm.; RV, 10 mm.; LV, 8 mm.
37.	C. C.	6 yr.	F	RVH.		Truncus arteriosus; pulmonary atresia; PDA, 2 mm.; IV septal defect 1.3 by 0.8 cm.; marked RVH.
38.	K. N.	5 mo.	M	LVH.		Coarctation of aorta; weight, 120 Gm.; RV, 2 to 3 mm.; LV. 13 mm.

IA = interauricular; IV = interventricular; LV = left ventricle; LVH = left ventricular hypertrophy; PDA = patent ductus arteriosus; RAD = right axis deviation; RBBB = right bundle branch block; RV = right ventricle; RVH = right ventricular hypertrophy.

B. *Other cyanotic patients:* Thirty-one patients in this group had unipolar electrocardiograms. The variety of lesions is tabulated in Table I. The electrocardiogram revealed right ventricular hypertrophy in twenty-five of these thirty-one cases and representative records are shown in Figs. 4 to 8. Six of the patients with cyanotic congenital heart disease had electrocardiograms revealing left ventricular hypertrophy. The lesions in these patients were as follows:

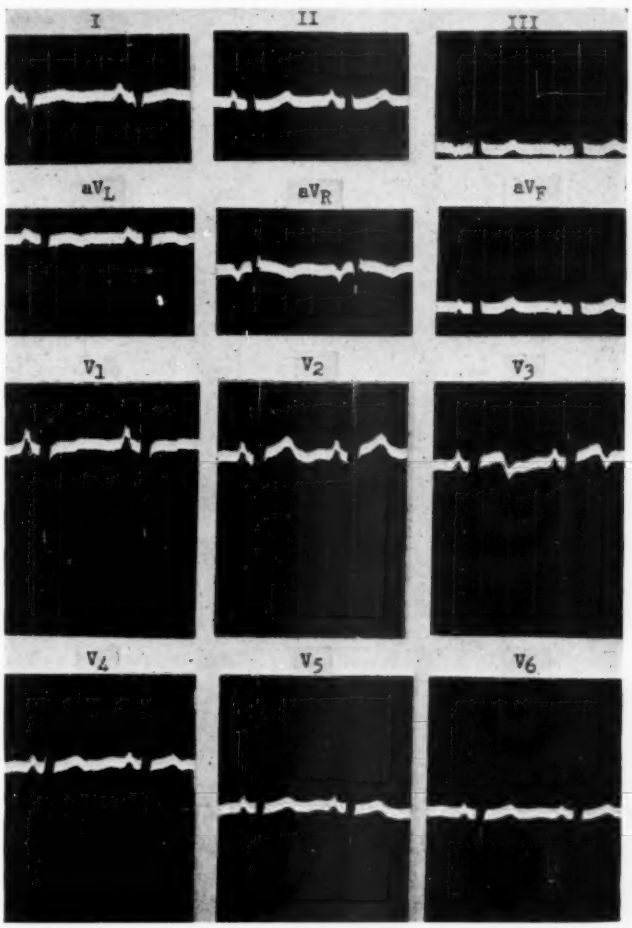

Fig. 5.—G. K., a boy aged 19 years, was cyanotic since birth. Autopsy revealed dextroposition of the aorta, pulmonary stenosis, I-V septal defect, large patent ductus supplying left lung, right ventricular hypertrophy (right ventricle 11 mm.), left ventricle measuring 13 mm. Electrocardiogram reveals right axis deviation but no definite evidence of either right or left vetricular hypertrophy.

tricuspid atresia with nonfunctioning right ventricle in three, tetralogy and large patent ductus arteriosus in two, truncus arteriosus and patent ductus arteriosus in one. Fig. 9 illustrates the findings in a patient with tricuspid atresia. Figs. 10 to 12 illustrate proved cases of right ventricular hypertrophy.

RELIABILITY OF THE CRITERIA OF VENTRICULAR HYPERTROPHY

In an attempt to determine the reliability of the criteria for diagnosis of left and right ventricular hypertrophy, a study was made of the thirty-eight patients in our series on whom autopsies were performed and on whom complete unipolar leads were taken. Table II summarizes the data on the thirty-eight cases. Of twenty-eight patients in this group diagnosed by means of the electrocardiogram as having right ventricular hypertrophy, all showed right ventricular hypertrophy at autopsy. Of the eight patients diagnosed as suggestive of or of having

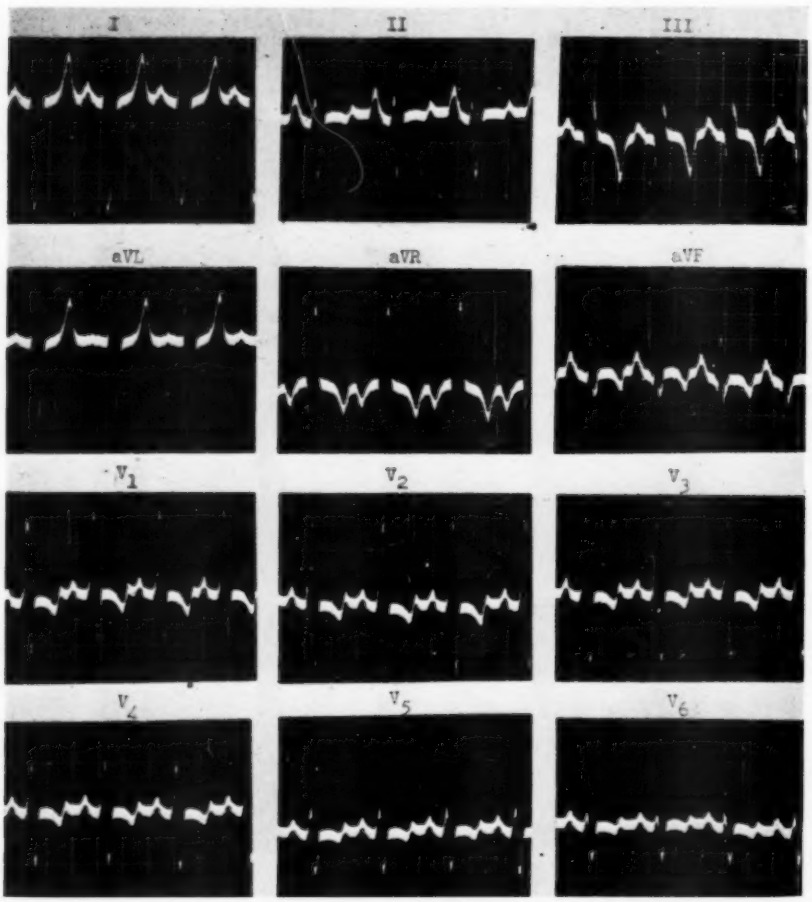

Fig. 6.—G. A. B., a female child aged 2 years, had transposition of the great vessels, absent interventricular septum, pulmonary stenosis, and patent foramen ovale (1 to 2 mm.), and hypertrophy of the right auricle (1.5 to 2.0 cm. thick). The single ventricle was 0.7 cm. thick.

definite left ventricular hypertrophy, all showed left ventricular hypertrophy at autopsy. Of the remaining two cases in which no diagnosis of ventricular hypertrophy was made, one (G. K., number 15 of the autopsy series), had a marked right axis deviation, but the electrocardiogram was not typical of either right

or left ventricular hypertrophy. Exploratory leads further to the right than V_1 and further to the left than V_6 might have been helpful but were not done.

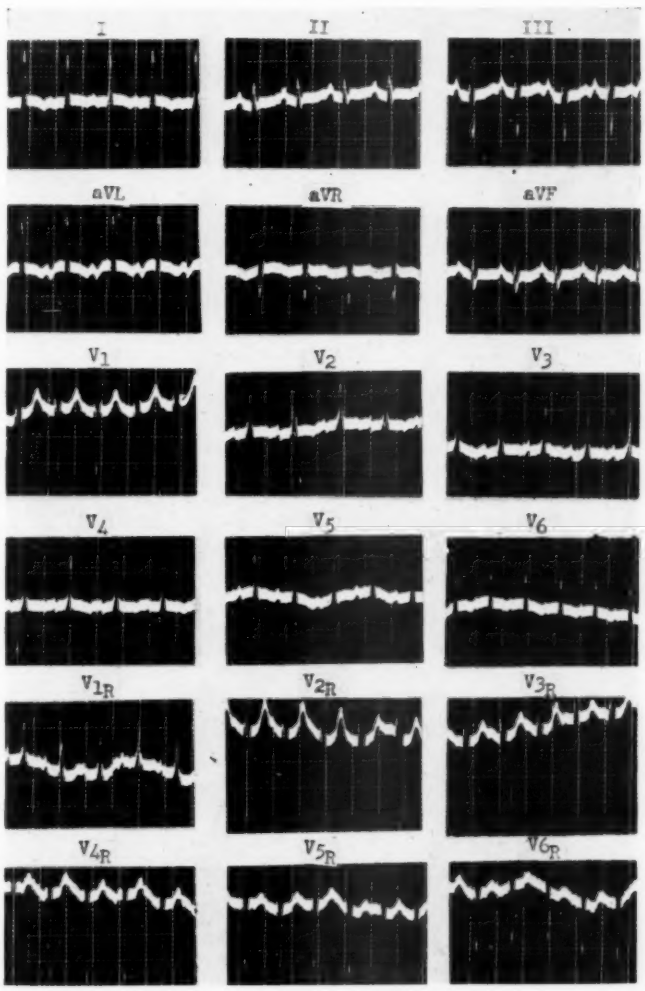

Fig. 7.—F. S., a female infant aged 3½ months, had progressive dyspnea and cyanosis. Autopsy (done elsewhere) revealed complete dextroposition of all organs and dextrocardia, nearly complete absence of the I-V septum, and right ventricular hypertrophy.

This patient had right ventricular hypertrophy at autopsy. The second patient (P. S., number 19 of the autopsy series) had an electrocardiogram that was probably within normal limits. At autopsy the heart was globular but the ventricular measurements were within the normal range. The patient was a 2-week-old infant.

It is apparent that the reliability of the diagnostic criteria is exceedingly high since no patient was diagnosed as having hypertrophy of either left or right

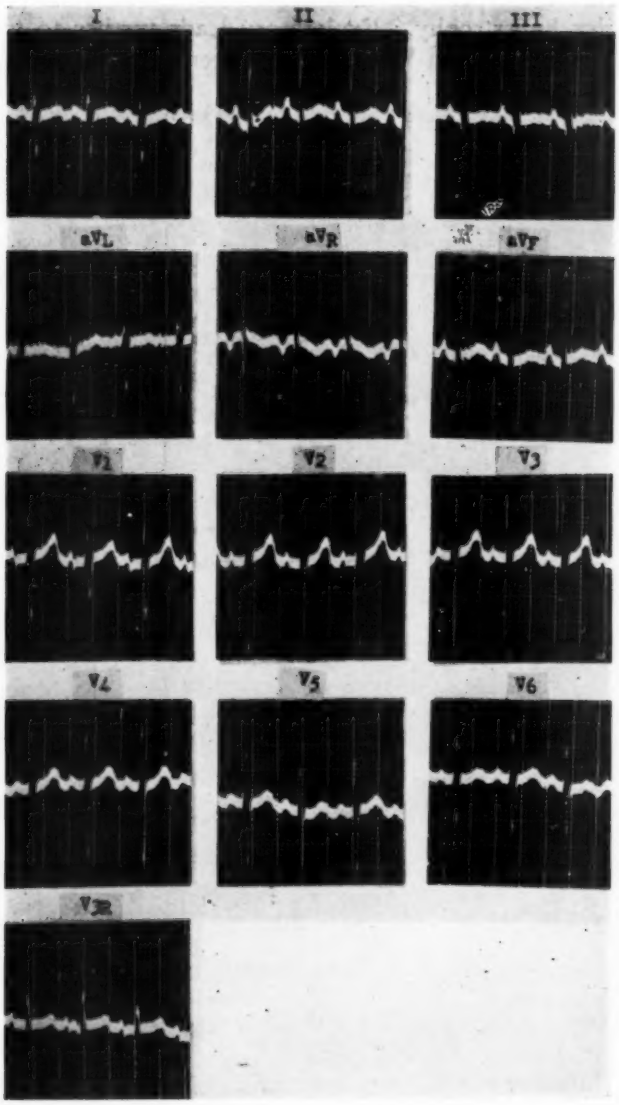

Fig. 8.—D. M. S., a female infant aged 4 months, had transposition of the great vessels, patent ductus arteriosus (3 mm. diameter), and interventricular septal defect (16 by 5 mm.). The right ventricle was 5 mm. thick. Monophasic R in V_{3R} suggests RVH but may be present normally in first few months of life. Transitional zone displaced to left, and V_6 does not reflect left ventricular potentials.

ventricle by means of the electrocardiogram who did not have such a finding at autopsy; in only one patient (G. K.) was major hypertrophy of a ventricle not diagnosed. This patient had marked right axis deviation but Lead V_1 was not characteristic of right ventricular hypertrophy. If exploratory leads over the right precordium had been taken, more accurate interpretation might have been possible.

DISCUSSION

The reliability of the electrocardiographic patterns of right and left ventricular hypertrophy seems established by the data presented in Table III. In only one case was there a major discrepancy (G. K.). In this patient there was a combined strain due to tetralogy of Fallot plus a patent ductus arteriosus. Despite a markedly hypertrophied right ventricle, the electrocardiogram showed

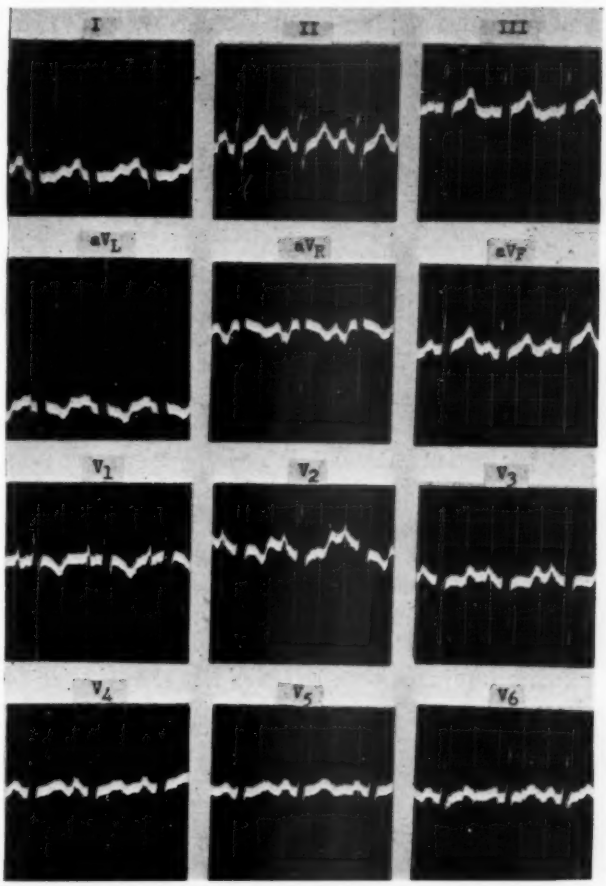

Fig. 9.—K. M. W., a girl child aged 2 years, had cyanosis, clubbing, mental and physical retardment since the age of 3 months. Systolic murmur and thrill were maximum in the left second and third interspaces. Cor triloculare or biloculare, large interauricular defect with endocardial band separating lower sixth of the atria were present. There was a small rudimentary right ventricle with a capacity of 5 c.c. communicating through a 3 mm. slit with a hypertrophied left ventricle (1 cm., 89 Gm.). The pulmonary artery was small, the aorta normal. There were joint mitral and tricuspid valves with wide openings.

no clear evidence of it, although the right axis deviation and prominent S wave in V_6 were suggestive. With this exception, significant hypertrophy was predictable when the typical electrocardiographic pattern was present. When the hypertrophy was minimal, a normal or borderline electrocardiogram was occa-

sionally found, indicating that errors in diagnosis may result from missing a minor lesion. When combined hypertrophy is present, the electrocardiogram usually reflects the changes in the ventricle with the major hypertrophy.

TABLE III. RELIABILITY OF CRITERIA OF VENTRICULAR HYPERTROPHY IN CONGENITAL HEART DISEASE AS DETERMINED AT AUTOPSY (38 CASES)

Right ventricular hypertrophy diagnosed electrocardiographically	28 cases	Right ventricular hypertrophy in all at autopsy
Left ventricular hypertrophy diagnosed electrocardiographically	8 cases	Left ventricular hypertrophy in all at autopsy
Right ventricular hypertrophy diagnosed at autopsy	29 cases	Right ventricular hypertrophy diagnosed electrocardiographically in 27 (Cases 15, 17)
Left ventricular hypertrophy diagnosed at autopsy	9 cases	Left ventricular hypertrophy diagnosed or suspected electrocardiographically in 8; combined hypertrophy in remaining case with preponderant left ventricular hypertrophy and electrocardiogram showed left ventricular hypertrophy (Case 2)

In the group of patients without cyanosis, the uncomplicated, and therefore surgically treatable, cases of coarctation of the aorta and patent ductus demonstrated either the pattern of left ventricular hypertrophy or were within normal limits. Of the six patients with either of these two lesions who, in retrospect, were inoperable because of associated lesions, four had right ventricular hypertrophy, one had questionable right ventricular hypertrophy, and one was within normal limits. The patient who had borderline right ventricular hypertrophy had coarctation of the aorta but also had a greatly enlarged heart and an intracardiac thrill. The patient with the normal record came to surgery with a preoperative diagnosis of coarctation but was found at surgery to have a hypoplastic arch and an intracardiac thrill. No patient with uncomplicated patent ductus arteriosus had an electrocardiographic pattern of right ventricular hypertrophy. It would appear, therefore, that in patients with coarctation of the aorta or patent ductus arteriosus, operation should be undertaken with great hesitancy in the presence of an electrocardiographic pattern of right ventricular hypertrophy because (1) the diagnosis may be in error, and (2) associated lesions may be present which would make surgery inadvisable.

The electrocardiographic pattern of right bundle branch block (complete or incomplete) occurred in three patients with interauricular septal defect, two patients with coarctation of the aorta, and in one patient with patent ductus arteriosus. The presence of the right ventricular conduction defect does not necessarily contraindicate surgery, since each of the last three patients was successfully operated upon.

The electrocardiogram was of value in providing some inference as to the diameter of the patent ductus arteriosus. In none of the patients with normal electrocardiograms was the ductus more than 1 cm. in diameter; one can predict, therefore, a small lesion in the presence of a normal electrocardiogram. In

contrast, many of the patients with the pattern of left ventricular hypertrophy had ducti of 1 cm. of more in diameter.

In the cyanotic group of patients with congenital heart disease, the majority had electrocardiographic patterns of right ventricular hypertrophy. This occurred, however, in the presence of a wide variety of lesions. In no patient

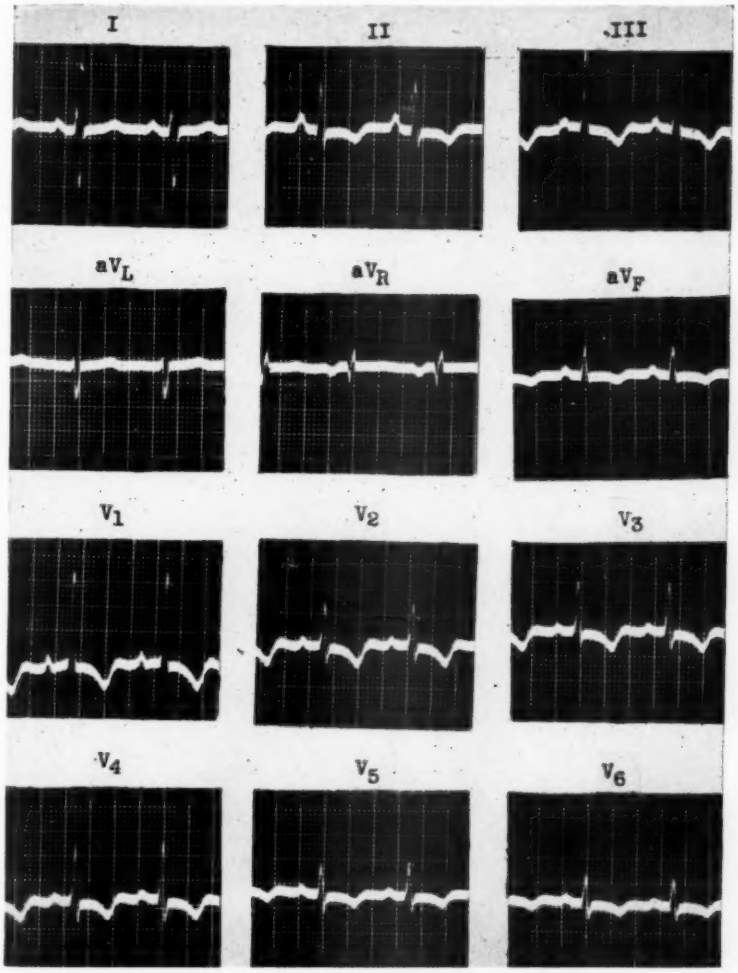

Fig. 10.—L. C., a woman aged 25 years, had pulmonary stenosis with patent interauricular septum, and cyanosis. The right ventricle was 1.5 cm.; the left ventricle, 1.1 cm. Coronary arteries were normal. Right ventricular hypertrophy was present.

over the age of 3½ months with tetralogy of Fallot was the electrocardiographic pattern of right ventricular hypertrophy absent. Left axis deviation with suggestive evidence of left ventricular hypertrophy in the presence of cyanotic congenital heart disease occurred in six patients. This combination has been described by Taussig[2,5] as being found almost exclusively in patients with tri-

cuspid atresia. In one of our six patients tricuspid atresia with nonfunctioning right ventricle and pulmonary stenosis was thought probable, and in two others it was proved at autopsy. In the remaining three patients, other lesions were present. In one, surgical exploration revealed an over-riding aorta with an anomalous patent ductus arteriosus. A second patient was found at operation to have a truncus arteriosus, patent ductus arteriosus, and an interventricular septal defect (P. E.). The axis deviation in this patient was minus 150 degrees.

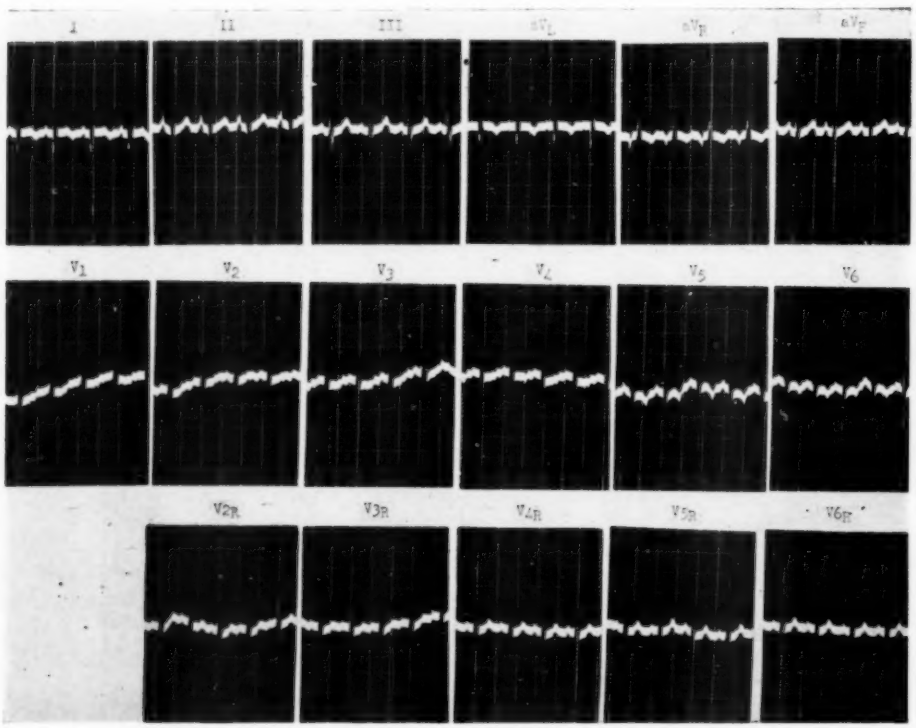

Fig. 11.—B. H., a female infant aged 17 weeks, had cyanosis since birth. Autopsy revealed cardiac enlargement with a right ventricle of 7 mm., a left ventricle of 5 mm., IV septal defect (1 cm. by 1 cm.), patent ductus arteriosus, and coarctation of the aorta. Death was due to cardiac failure, pneumonia, and atelectasis. Note early electrocardiographic signs of right ventricular hypertrophy confined to the left precordial leads with a small R/S ratio.

A third patient was found at operation to have an interauricular septal defect, patent ductus arteriosus, and tetralogy. The axis deviation was minus 30 degrees. Therefore, only three of the six patients with cyanotic congenital heart disease in which left ventricular hypertrophy and not right ventricular hypertrophy was suggested by the electrocardiographic changes had the syndrome of tricuspid atresia with nonfunctioning right ventricle.

The reliability of the criteria of ventricular hypertrophy was quite striking; there were no false positives and there was almost complete diagnostic accuracy (as far as the chamber showing hypertrophy was concerned) in the autopsy cases. Only one major error, that of overlooking conspicuous hypertrophy of a

ventricle, occurred. When combined hypertrophy is present, the electrocardiogram may reveal the preponderant hypertrophy and the fact that the opposite ventricle is enlarged may be overlooked.

In one instance the electrocardiogram revealed right ventricular hypertrophy, whereas no right ventricle could be seen following Diodrast angiocardiogram. At autopsy, the patient had pulmonary atresia with conspicuous right ventricular hypertrophy but with a very small right ventricular cavity.

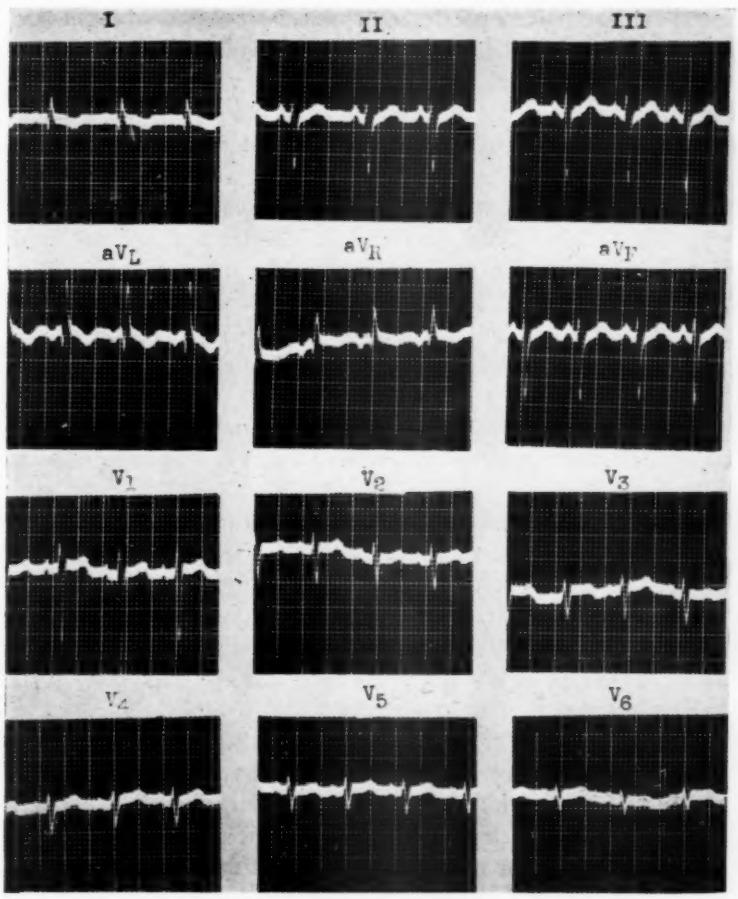

Fig. 12.—R. C., a male infant aged 10 months, had hypoplastic pulmonary artery, interauricular septal defect, dextrocardia, and right ventricular hypertrophy confirmed at autopsy.

In the cases with electrocardiographic evidence of right ventricular hypertrophy in whom cardiac catheterization was performed (by Dr. Sanford Leeds) (six cases), all had mean right ventricular pressures exceeding 35 cm. saline.

The relative value of the unipolar electrocardiogram and radiologic techniques in the recognition of ventricular hypertrophy is to be studied further.

SUMMARY AND CONCLUSIONS

1. The electrocardiographic findings utilizing unipolar precordial and extremity leads in 153 patients with congenital cardiac disease have been presented.

2. Of thirty-nine patients with uncomplicated patent ductus arteriosus, unipolar electrocardiograms revealed left ventricular hypertrophy in seventeen, incomplete right bundle branch block in one, and were within normal limits in twenty-one. None of the patients with uncomplicated patent ductus arteriosus had right ventricular hypertrophy. Seven additional patients with patent ductus had associated cardiac lesions producing cyanosis.

3. A normal electrocardiogram in the presence of uncomplicated patent ductus arteriosus was associated in all cases with a ductus not exceeding 1 cm. in diameter. All the patients in whom the diameter of the ductus exceeded 1 cm. revealed electrocardiographic patterns of left ventricular hypertrophy.

4. Of thirty-two patients with coarctation of the aorta, nineteen had electrocardiographic patterns of left ventricular hypertrophy, two had right ventricular conduction defects, six had normal electrocardiograms, and five had electrocardiographic patterns of right ventricular hypertrophy. The five patients in whom right ventricular hypertrophy was found had associated lesions or cardiac failure with a patent foramen ovale. Five of the acyanotic patients had associated congenital defects. In two additional patients coarctation was associated with a lesion producing cyanosis.

5. In eight cases of interauricular septal defect, the electrocardiogram revealed right ventricular hypertrophy in five and incomplete right bundle branch block in three.

6. In forty cases of tetralogy of Fallot, all patients showed right ventricular hypertrophy except one, aged $3\frac{1}{2}$ months, in whom the electrocardiogram suggested right bundle branch block more than right ventricular hypertrophy. The electrocardiographic pattern of right ventricular hypertrophy was found in a wide variety of cyanotic cardiac lesions and therefore was not of specific diagnostic value. In no case of tetralogy of Fallot was the electrocardiogram normal nor did it show left ventricular hypertrophy.

7. In six of the cyanotic cardiac patients the electrocardiogram revealed left axis deviation or left ventricular hypertrophy. In four patients the precordial patterns suggested left ventricular hypertrophy, but in the other two they were not typical of either right or left ventricular hypertrophy. Two of these six patients had tricuspid atresia with a nonfunctioning right ventricle and pulmonary stenosis, and a third probably had this lesion.

8. In five patients with transposition of the great vessels, all showed electrocardiographic evidence of right ventricular hypertrophy. In two of these five, the pattern of right ventricular hypertrophy was not fully developed.

9. The diagnostic accuracy of the criteria employed in the recognition of right and left ventricular hypertrophy is shown, and the value of the knowledge of such hypertrophy in the diagnosis of congenital heart disease is presented.

We are grateful to the Divisions of Pediatrics, Radiology, Thoracic Surgery, and Pathology for their cooperation.

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AN ELECTROCARDIOGRAPHIC STUDY OF PSYCHIATRIC PATIENTS BEFORE AND AFTER ELECTROSHOCK THERAPY

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THERE is a widespread belief that electroshock treatment as used in the treatment of psychiatric patients places a severe strain on the cardiovascular system.[1-3] Therefore, where cardiac consultation is not available, great reliance is placed on electrocardiography in evaluation of risk in the treatment of such cases. In most clinics this is routine. The majority of these patients are presented to the psychiatrist as purely mental cases and in many instances a medical history cannot be obtained. There are always some patients, however, who have cardiovascular disease complicated by psychiatric disorders.[3] In the course of this routine electrocardiographic study we were impressed by the number of abnormalities uncovered, a number which was far in excess of what might have been anticipated in routine study of unselected patients.

At first we were extremely cautious about approving shock treatment in the presence of T-wave changes which seemed to indicate myocardial injury. In a number of cases the necessity of doing something to improve the psychiatric state outweighed the presumed risk, and the treatment was given uneventfully. In a great many instances in our series, electrocardiograms made after the completion of electroshock therapy showed the changes previously present to have disappeared. This had been shown to occur in an occasional case by Winton and Wallace.[23]

It is vital to remember that electrocardiographic changes may be produced by extracardiac as well as cardiac causes, because otherwise many patients will be denied treatment. As a matter of fact, the causes of changes in the T waves and S-T sectors are legion. Some of the well-known causes are avitaminosis,[4] hot and cold applications,[6,7] hot and cold drinks,[5] drugs,[6,8-10] position of the body,[8,9,11] abdominal distentions,[12] hyperadrenalemia,[15] emotions,[19,20] hyperventilation,[13] neurocirculatory asthenia,[9,18,21,23] fever,[16] exercise,[11] orthostatic hypotension,[8] postoperative disturbances,[6] hyper- and hypopotassemia,[16,18] and other electrolyte disturbances.[11,13,14,16]

It is less well known that autonomic imbalance[9,10,15,22-24] and the psychoses[22,25-27] influence the electrocardiogram. It has been shown, for example, that vagotonia often results in tall T waves and sympathicotonia produces low, diphasic or inverted T waves.[22] Acidosis mimics the former and alkalosis the latter.[11]

In an effort to throw more light on the relationship of psychiatric disturbances to electrocardiographic abnormalities, we studied tracings of fifty unselected

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patients from the neuropsychiatric unit. One or more electrocardiograms were made before treatment and one at the time of discharge. Included in these 50 cases were: 2 paranoid psychoses, 8 senile and presenile psychoses, 3 manic-

TABLE I. ELECTROCARDIOGRAMS AFTER ELECTROSHOCK

PSYCHIATRIC DIAGNOSIS	IMPROVED	UNCHANGED	WORSE
2 paranoid psychoses	2	0	0
1 barbiturate addiction	0	1	0
4 senile psychoses	1	2	1
3 manic depressives	1	2	0
4 presenile agitated depressions	1	1*	2
19 involutional psychoses	11	8	0
8 schizophrenias	4	1*	3
7 agitated depressions	3	3*	1
2 psychoneuroses	1	1	0

*Cases were among the abnormal before electroshock for organic reasons.

TABLE II. ELECTROCARDIOGRAPHIC CHANGES

	BEFORE ELECTROSHOCK	AFTER ELECTROSHOCK
PR Interval		
Unchanged	45	45
Prolonged	2	3
Decreased	3	2
T Wave in Lead I		
Less than 0.5 mm.	16	4*†
Diphasic	4	3*†
Inverted	4	3*
T Wave in Lead II		
Less than 0.5 mm.	4	0
Diphasic	3	0
Inverted	4	3*
Depressed ST-J in Leads I and II	9	2*†
Diphasic Precordial T Waves		
CR	7	1*
V	6	4*†
Low Precordial T Waves		
CR	6	1†
V	8	2*†
Depressed Precordial ST-J		
CR	7	0
V	4	0
Inverted Precordial T Waves	5	2*
Auricular Fibrillation	1	1*

*Nineteen electrocardiograms were abnormal before electroshock and were unchanged. Five of these were due to organic leasions.

†Seven electrocardiograms became abnormal after electroshock.

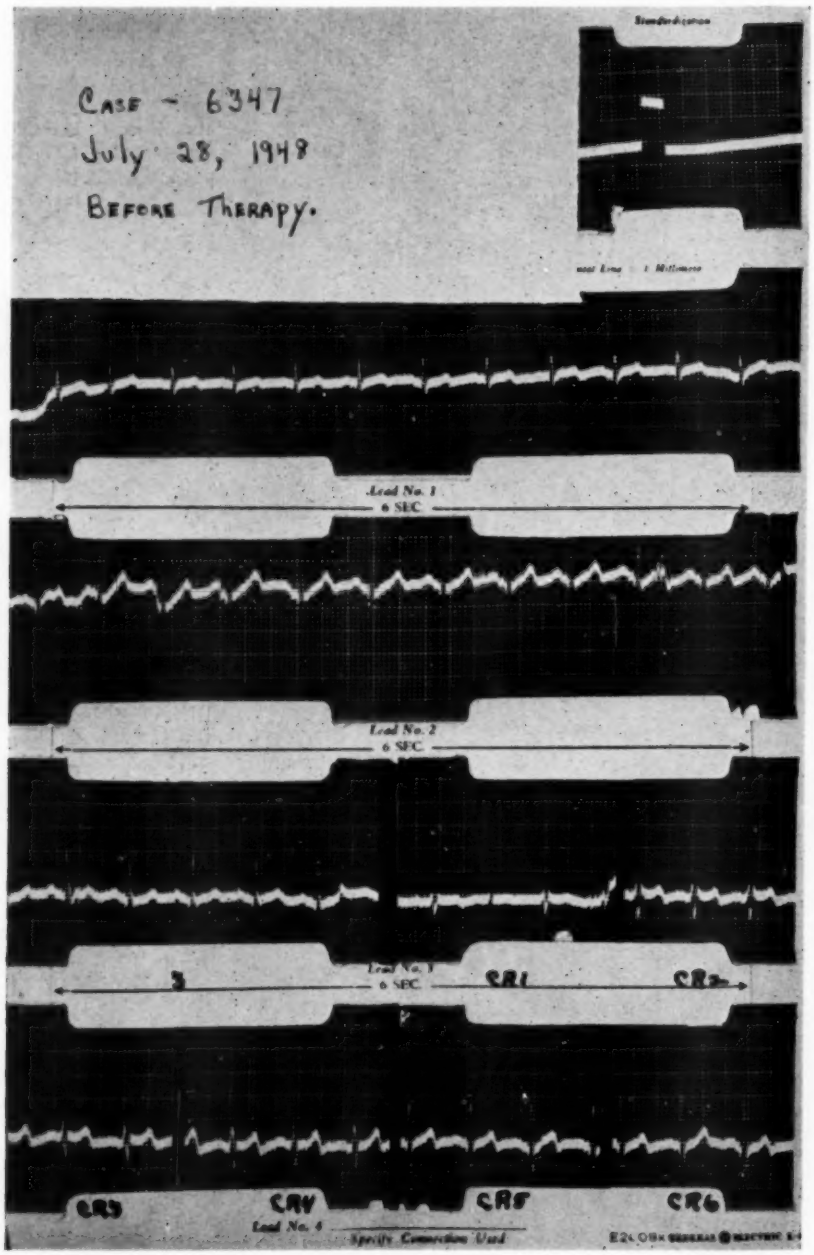

Fig. 1A.

depressives, 19 involutional psychoses, 8 schizophrenias, 7 agitated depressions, 2 psychoneuroses, 1 barbiturate addiction (Table I). The ages of these patients varied from 21 to 76 years. There were eighteen men and thirty-two women. Five patients had definite electrocardiographic abnormalities, including two with

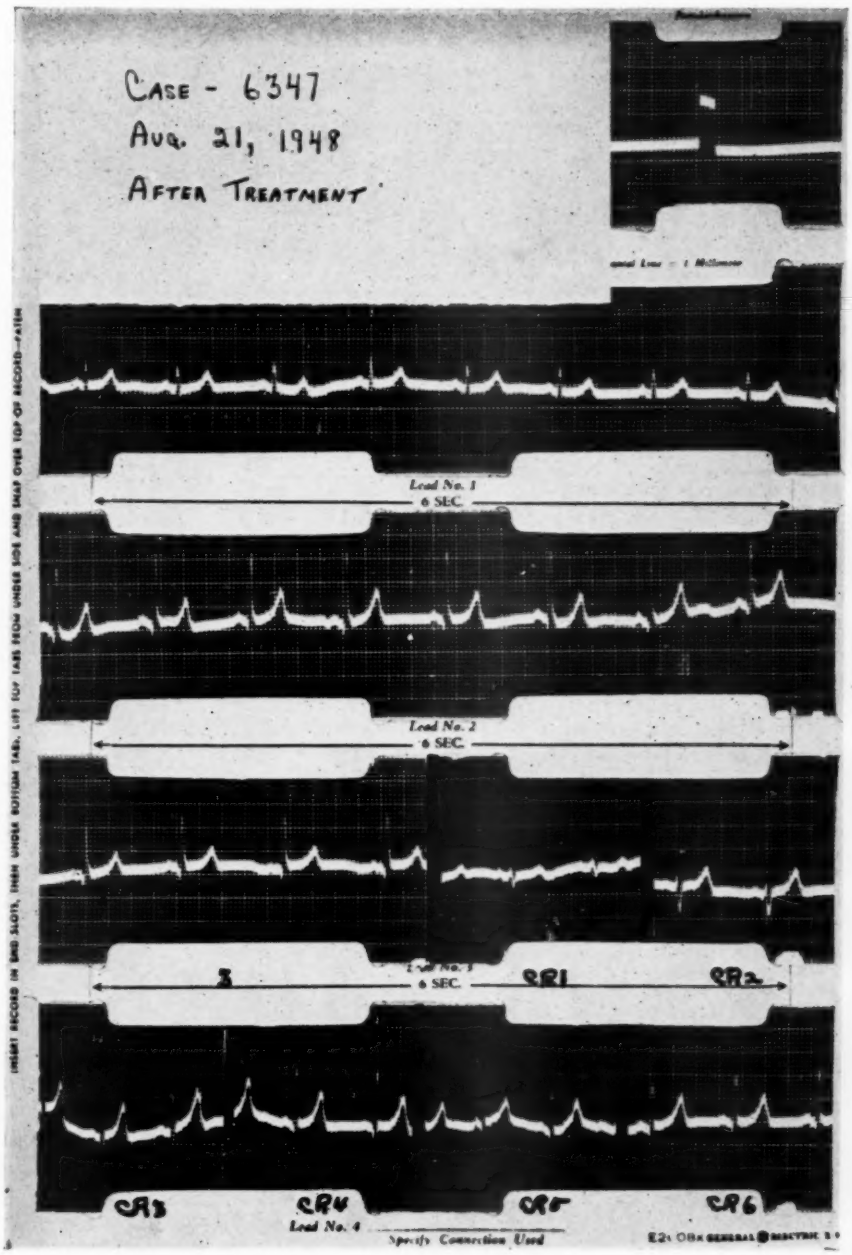

Fig. 1B.

left ventricular hypertrophy, one with healed anterior infarct, one with healed posterior infarct, and one with congenital dextrocardia.

We found the T waves in Lead I to be less than 0.5 mm. in amplitude in sixteen cases before treatment and in only four cases after treatment. T waves

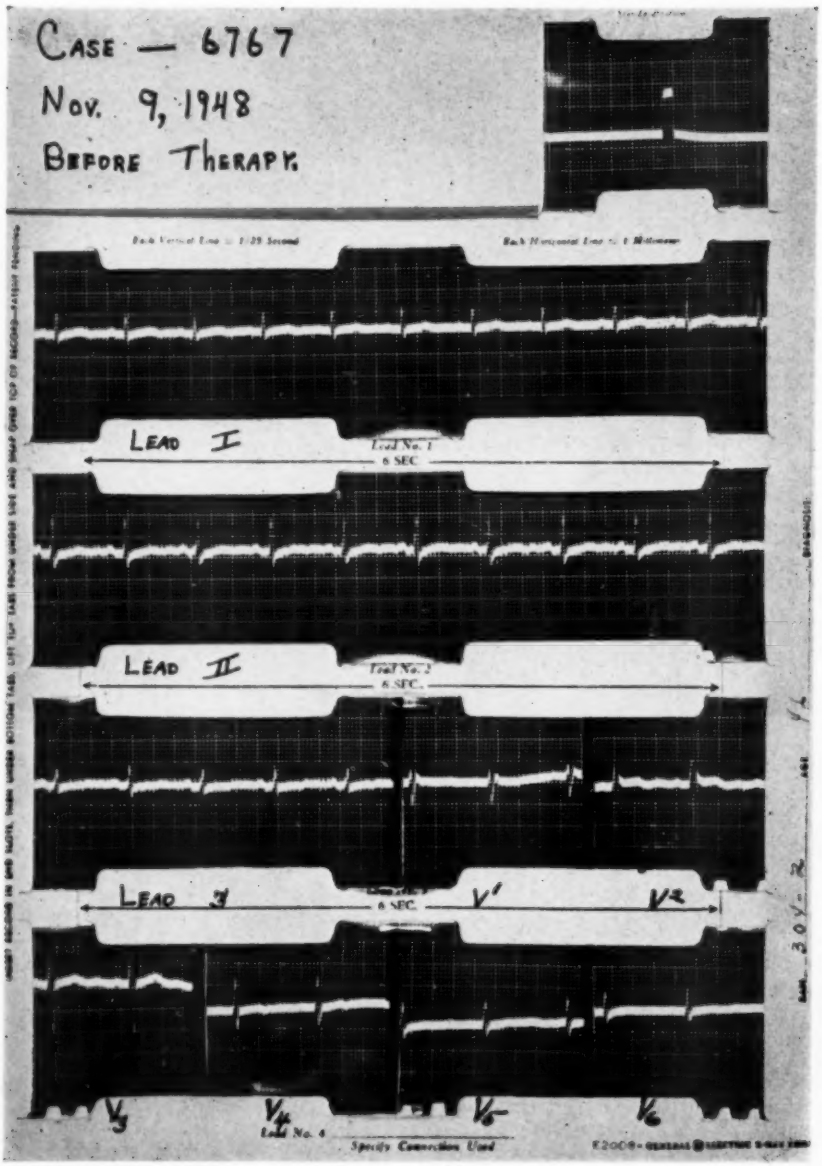

Fig. 2A.

less than 0.5 mm. in Lead II occurred in four cases before treatment but were all normal after treatment. The same was true of three cases with diphasic T waves in Lead II. Of four patients with inverted T waves in Leads I and II, only one returned to normal, the other three being abnormal for organic reasons. Of nine patients with depressed S-T sectors in Leads I and II seven were normal after treatment. Diphasic T waves in the precordial leads, either CR or V, were found in thirteen or 26 per cent before treatment, and returned to normal in

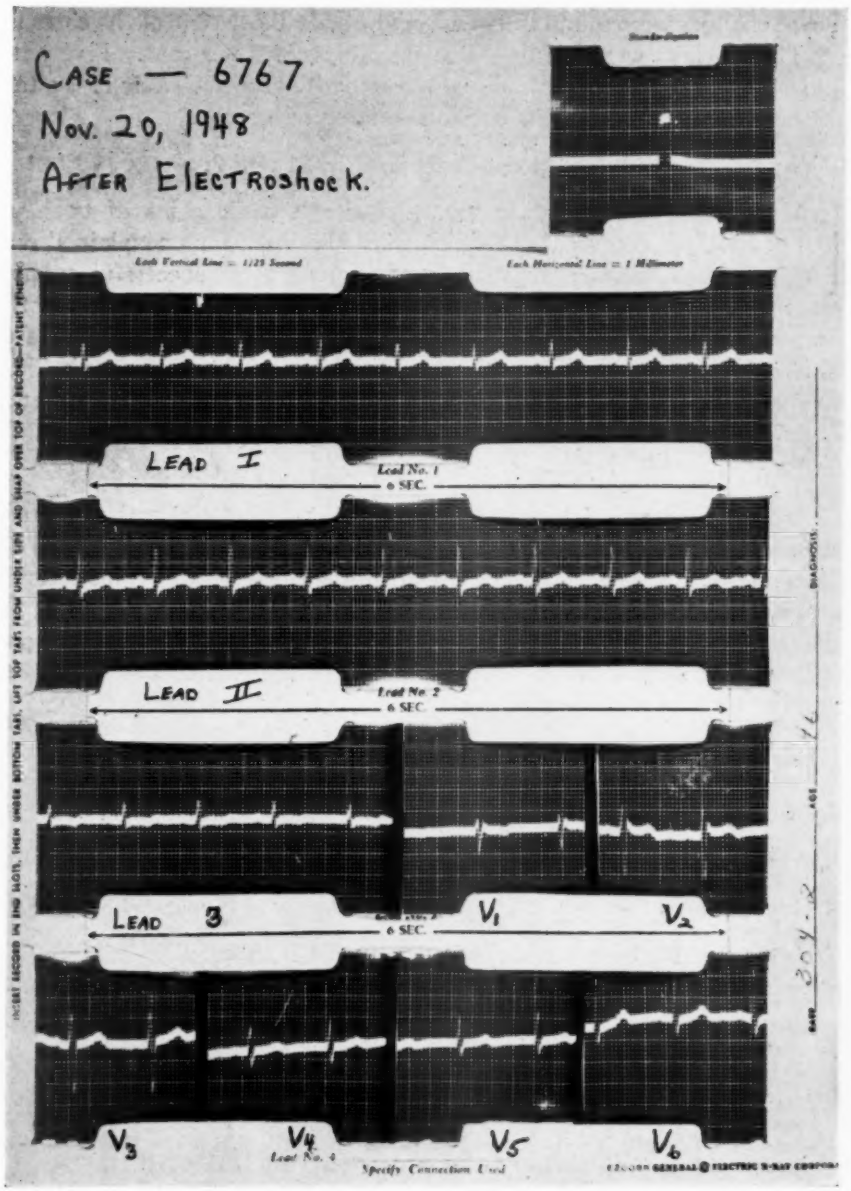

Fig. 2B.

eleven or 84.6 per cent after treatment. Low T waves in precordial leads were found in fourteen or 28 per cent and returned to normal in eleven or 78.6 per cent after treatment. Depressed S-T sectors occurred in the precordial leads in eleven or 22 per cent and returned to normal in all. Inverted T waves were found in precordial leads in five or 10 per cent and returned to normal in three or 60 per cent.

These changes were found in twenty-eight or 56.0 per cent of the cases in our series. The tracings were normal in seventeen or 34.0 per cent. Five patients with known heart disease had abnormal electrocardiograms. Table II is a tabulation of these findings in all cases before and after treatment.

Typical electrocardiograms are shown in Figs. 1 and 2.

It is evident from our data that the electrocardiograms of twenty-four patients, or 85.7 per cent of those which were abnormal for unexplained reasons before electroshock, showed marked improvement after successful treatment. It seems fair to assume that the electrocardiographic abnormalities must have been in some way related to the mental state because they are otherwise unexplainable. We are of the opinion that the mechanism of these changes in T waves and S-T sectors is through the operation of autonomic nervous system disturbances.

CONCLUSION

Electrocardiographic changes occurring in psychiatric patients must be differentiated from those due to other extracardiac causes as well as those due to associated cardiac disease. The majority of these electrocardiograms returned to normal after electroshock treatment and rehabilitation of these patients. Such changes should not be interpreted as due to organic cardiac lesions and the patient denied treatment on that basis. In our practice no patient has been denied treatment on the basis of T-wave changes.

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MULTIPLE LEAD ELECTROCARDIOGRAMS IN ANGINA PECTORIS

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THE increased general use of unipolar limb and multiple precordial leads makes it desirable to re-evaluate the role of electrocardiography in medical practice. In the present study the twelve lead electrocardiograms (three standard, three augmented unipolar limb, and six unipolar precordial leads) of patients with uncomplicated angina pectoris are compared with those of a group of subjects similar in age but without clinical symptoms or signs of heart disease. The purpose is to determine what such tracings show in patients with angina pectoris and to differentiate these findings from those of patients with acute myocardial infarction on the one hand and those with no symptoms or signs of heart disease on the other.

The changes in Q, RS-T, and T characteristic of acute myocardial infarction are well known and are of considerable diagnostic importance when well defined and progressive. When such findings are borderline, however, their significance is difficult to evaluate. For example, small Q waves, slight elevation or depression of RS-T segments, or low amplitude T waves may represent: (1) the earliest objective evidence of acute myocardial infarction; (2) the only residual evidence of past infarction; (3) myocardial disease without infarction; or (4) they may occur in the absence of myocardial damage.

The limits and variations of normal in unipolar limb and precordial electrocardiograms have not been established with certainty, and the frequency of borderline tracings in apparently normal adults is not well known. Until sufficient experience has accumulated to be certain of these fundamentals, it is possible that the significance of minor changes may be overemphasized. Not uncommonly, unipolar limb and multiple precordial leads are obtained only when cardiac disease is suspected. This may result in inadequate experience with the normal variations.

MATERIAL AND METHODS

Angina Pectoris Patients.—The records of 134 patients with angina pectoris but no other condition which might influence the electrocardiogram were used for this study. These patients had all been studied in the Beth Israel Hospital of Boston in 1947 and 1948. Approximately 30 per cent had been admitted for treatment of their attacks; the remaining 70 per cent were admitted for reasons unrelated to angina pectoris. The 134 patients could be divided into two groups:

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Group I included 100 patients from whom no past history of myocardial infarction could be obtained; Group II consisted of thirty-four patients with a past history of myocardial infarction.

During the same period there were 280 additional patients with angina pectoris who were not suitable for this study. These included eighty-seven with inadequate histories or inadequate electrocardiographic studies, ninety-eight with recent (within six months) myocardial infarction or progression of symptoms suggesting acute myocardial damage, sixty-one receiving digitalis, thirty with valvular heart disease, one with proved ventricular aneurysm, two with anemia, and one with myxedema.

Control Subjects.—A third group of 100 subjects all 45 years of age or over who had neither clinical symptoms nor signs of heart disease served for comparison. Because of the difficulty of finding such subjects among hospitalized patients, these were obtained from the private practice of Drs. Harry Linenthal and Joseph E. F. Riseman. The medical histories of these subjects were well known, for they had made it their custom to have periodic annual or semi-annual examinations for two to twenty years or more. Multiple lead tracings were obtained in 1948 and 1949 as part of such a periodic examination. All were living normally active lives and were free from major complaints.

Clinical Characteristics.—The age distribution of the three groups is shown in Table I; the distribution according to sex, blood pressure, and heart size is shown in Table II. In most instances the heart size was determined by physical examination only.

TABLE I. AGE DISTRIBUTION OF 100 CONTROL SUBJECTS AND 134 PATIENTS WITH ANGINA PECTORIS

AGE	SUBJECTS WITHOUT CARDIAC SYMPTOMS (% OF 100 NORMAL SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (% OF 100 PATIENTS)	PAST HISTORY OF INFARCTION (% OF 34 PATIENTS)
34 to 39 years	0	4	6
40 to 49 years	19*	9	9
50 to 59 years	40	29	35
60 to 69 years	32	36	44
70 to 79 years	9	20	6
80 to 89 years	0	2	0

*Four subjects were 45 years of age; all others were older.

Electrocardiograms.—The electrocardiograms were all taken with the subjects at rest in the supine position. Three bipolar standard leads (I, II, III), three augmented unipolar limb leads (aV_L, aV_R, aV_F), and six unipolar precordial leads (V_1 to V_6) were obtained in all instances. Vacuum tube amplifier photographic electrocardiographs (Sanborn Cardiette) and the Wilson unipolar terminal were used in all instances.

Because of lack of complete agreement as to the limitations of normal, the borderline or questionably normal tracings are presented separately (see Table

V). The undoubtedly normal tracings showed regular rhythm (except for inconstant premature beats), no conduction defects, Q waves which were absent or small, RS-T segments which were elevated or depressed less than 0.5 mm. in standard or limb leads and less than 1 mm. in precordial leads, and T waves which were upright and more than 1.5 mm. in Leads I and II and 1 mm. or more in Leads $V_{4,5,6}$. The direction of T in Leads aV_L and aV_F was considered in relation to other characteristics in the tracings, for as Goldberger has reported[1] the normal T wave may vary from plus 4 to minus 2.5 mm. in aV_L and from plus 5 to minus 1 mm. in aV_F.

TABLE II. CLINICAL CHARACTERISTICS OF 234 SUBJECTS

	SUBJECTS WITHOUT CARDIAC SYMPTOMS (% OF 100 SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (% OF 100 PATIENTS)	PAST HISTORY OF INFARCTION (% OF 34 PATIENTS)
Sex			
Men	70	60	74
Women	30	40	26
Heart Size			
Normal	100	51	47
Enlarged	0	49	53
Blood Pressure (mm. Hg)			
100/60 to 138/84	35	28	29
140/90 to 160/99	35	34	35
165/100 to 250/140	30	38	36

RESULTS

The subjects without cardiac symptoms showed normal electrocardiograms in 85 per cent and abnormal tracings in the remaining 15 per cent (Table III). The angina patients without a history of past myocardial infarction had normal tracings in 52 per cent and abnormal tracings in 48 per cent, while those with a history of infarction had normal tracings in 21 per cent and abnormal tracings in 79 per cent. Despite the difference in the frequency of abnormal tracings, there was no difference in the frequency and severity of the attacks in these two groups of patients with angina.

In the present series, there were no significant differences between the electrocardiograms of patients admitted because of their angina pectoris and those who came to the hospital for other reasons. There were no important differences between the tracings of the men and those of the women. No constant relationship was found between the electrocardiogram and the heart size or the blood pressure. The position of the heart as indicated by the unipolar limb leads (Table IV) revealed a somewhat higher incidence of horizontal hearts in patients with angina pectoris than in those without cardiac symptoms. Semivertical or vertical hearts were relatively infrequent in all three groups.

TABLE III. FREQUENCY OF NORMAL AND ABNORMAL ELECTROCARDIOGRAMS IN 100 CONTROL SUBJECTS AND 134 PATIENTS WITH ANGINA PECTORIS

	SUBJECTS WITHOUT CARDIAC SYMPTOMS (% OF 100 SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (% OF 100 PATIENTS)	PAST HISTORY OF INFARCTION (% OF 34 PATIENTS)
Normal	85	52	21
Abnormal	15	48	79

Normal Electrocardiograms.—In most instances these tracings were unquestionably normal by all accepted standards. In about one-third of the normal tracings of the control subjects and one-half of the normal tracings of the angina subjects, the characteristics of Q, RS-T, and T in one or more of the twelve leads have been considered by some standards to indicate myocardial damage (Table V). For example, deep Q waves in Lead III, especially if accompanied by inversion of T_{III}, or T waves in Lead I which are approximately 1 mm. in amplitude have been considered by some insurance companies as sufficient evidence to warrant refusal of life insurance. The unipolar limb leads are of particular value in determining when such findings are due to myocardial damage and when they are the result of the cardiac position. Unfortunately, only the three standard leads and $V_{2,4,5}$ are obtained routinely for insurance purposes.

In the present series, the six instances of deep Q waves in Lead III were unassociated with any abnormalities in the twelve lead tracings. The absence of Q waves in aV_F indicates that these prominent Q_{III} waves were not due to previous myocardial infarction. Similarly, there were twenty-three instances where T_I was upright but 1 mm. or slightly less in amplitude, the remainder of the twelve lead tracings being normal. In twenty-one of these, T_{II} was greater in amplitude and T_{III} was upright. The aV_L leads in these twenty-three instances showed T to be upright but 1 mm. or less in amplitude in seventeen, isoelectric in three, and minus 0.5 to 1 mm. in the remaining three. Lead aV_R showed T to be inverted in all twenty-three instances. These measurements in aV_L and aV_R are within the limits of normal as given by Goldberger.[1]

TABLE IV. ELECTROCARDIOGRAPHIC POSITION OF THE HEART IN 234 SUBJECTS

	SUBJECTS WITHOUT CARDIAC SYMPTOMS (% OF 100 SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (% OF 100 CASES)	PAST HISTORY OF INFARCTION (% OF 34 CASES)
Horizontal	25	38	44
Semihorizontal	22	20	15
Intermediate	37	37	32
Semivertical	11	3	0
Vertical	5	2	9

TABLE V. "BORDERLINE" OR "SUSPICIOUS" ELECTROCARDIOGRAMS REGARDED AS WITHIN THE RANGE OF NORMAL

	SUBJECTS WITHOUT CARDIAC SYMPTOMS (NO. SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (NO. PATIENTS)	PAST HISTORY OF INFARCTION (NO. PATIENTS)
Deep Q waves in Lead III	6	0	0
QS waves in V_1	3	0	0
RS-T elevated 1 to 3 mm. in V_1 to V_3 or V_2 to V_4	6	7	2
T_1 upright, 0.5 to 1 mm. in amplitude	6	10	2
T in $V_{4,5,6}$ upright but less than 1 mm.	3	4	0
T_1 low in amplitude plus QS waves in V_1	1	0	1
T_1 low in amplitude plus RS-T elevated in $V_{1,2,3}$	1	2	0
Right bundle branch block	3	3	1
Total	29	26	6

Elevation of RS-T segments or deep QS waves in leads from the right side of the heart have been considered evidence of myocardial damage especially if, clinically, there is a probability of recent cardiac injury. It is evident, however, that such findings may occur in the absence of any clinical evidence of damage and are of no diagnostic significance if they are the only unusual findings in the twelve lead tracings. In such instances, serial tracings are necessary to prove that these are not progressive changes.

In the present study, T waves which were absent, diphasic, or inverted in $V_{4\ and\ 5\ and\ 6}$ were invariably associated with other electrocardiographic evidence of abnormality. In seventeen patients with angina pectoris, T in V_4 was upright (1 to 3 mm.) while T in $V_{5,6}$ was diphasic or inverted; in all these instances there were other abnormalities elsewhere in the twelve lead tracings. Thus it appears that $V_{5,6}$ may reveal abnormalities of T not evident in V_4 or a single apical precordial lead.

The T wave was upright but less than 1 mm. in amplitude in $V_{4,5,6}$ in three of the control subjects and in four angina patients; the twelve lead tracings were otherwise normal. These T waves are considerably less in amplitude than the lower limits of normal as given by the Criteria Committee of the New York Heart Association.[2] Many investigators, however, have considered the T wave in leads from the left side of the heart to be abnormal only if it is absent or inverted.[3-9]

Right bundle branch block was found in three control subjects and in four angina patients. It is generally agreed that the prognosis of such patients is excellent (if there is no other evidence of cardiac damage). They are included among the "borderline" or "suspicious" tracings because of the rarity of their occurrence in young people.

Abnormal Electrocardiograms.—Abnormal electrocardiograms were found in 15 per cent of the control subjects, 48 per cent of angina patients without a history of past infarction, and 79 per cent of angina patients with past infarction (Table VI). In almost all instances multiple abnormalities were present; the combinations, however, varied considerably, and the commonly recognized patterns were in the minority.

TABLE VI. ABNORMAL ELECTROCARDIOGRAMS

ABNORMALITY	SUBJECTS WITHOUT CARDIAC SYMPTOMS (NO. SUBJECTS)	PATIENTS WITH ANGINA PECTORIS	
		NO HISTORY OF INFARCTION (NO. PATIENTS)	PAST HISTORY OF INFARCTION (NO. PATIENTS)
Deep Q waves in aV_L	1	3	4
Plus inverted T waves in aV_L	1	1	1
Plus inverted T waves in Lead I	0	0	1
Plus inverted T waves in Leads I and $V_{4,5,6}$	0	2	1*
Plus inverted T_I and QS waves in $V_{2,4,5}$	0	0	1
Deep Q waves in aV_F	4	6	10
Plus inverted T waves in aV_F	1	0	2
Plus inverted T waves in Lead I	0	2*	2
Plus inverted T waves in Leads $V_{4,5,6}$	3	1	4
Plus inverted T waves in Leads I and $V_{4,5,6}$	0	3†	2
RS-T Elevated in Leads I, II, III, aV_L, and V_1 to V_6 but Depressed in aV_R	1	0	0
T Waves Inverted or Absent in Lead I	2	0	0
T Waves Inverted or Absent in $V_{4,5,6}$	2	3	4
No other abnormality	1	0	1
Plus RS-T elevated in $V_{1,2,3}$	1	3	3
T Waves Inverted or Absent in Leads I and $V_{4,5,6}$	3	32	8
No other abnormality	1	19	2
Plus QS waves in V_1	1	3	0
Plus QS waves in $V_{1,2,3}$	0	1*	5†
Plus elevated RS-T in $V_{1,2,3}$	1	0	0
Plus depressed RS-T in $V_{4,5,6}$	0	9	1
Left Bundle Branch Block	0	1	0
P-R Intervals 0.24 to 0.26 Second	2	1	1
Auricular Flutter	0	1	0
Auricular Fibrillation	0	1	0
Total	15	48	27

*These patients also had RS-T segment elevation in $V_{1,2,3}$.
†All but one of these patients also had RS-T segment elevation in $V_{1,2,3}$.

Inverted, diphasic, or absent T waves in Leads I and $V_{4,5,6}$ was the combination most frequently encountered. This occurred in three of the fifteen abnormal tracings of the controls (20 per cent) and in forty-eight of the seventy-five abnormal tracings of the angina patients (64 per cent). In most instances these T wave changes were accompanied by QS waves in $V_{1,2,3}$, elevation of RS-T in $V_{1,2,3}$, depression of RS-T in $V_{4,5,6}$, or Q waves in the augmented limb leads. These T-wave changes in Leads I and $V_{4,5,6}$ were part of the electrocardiographic pattern of left ventricular hypertrophy (left axis deviation plus inverted T_I in the standard leads; horizontal heart plus inversion of T in aV_L in the limb leads; and prominent, though not abnormally high, R waves plus inversion of T in V_4) in two of the three control subjects and nine of the forty-eight angina patients. None of the three control subjects but six of the nine angina patients showed cardiac enlargement on physical examination.

There were two patients (each with angina pectoris and a past history of infarction) who showed T waves more than 10 mm. in amplitude in leads from the left side of the chest (measuring 14 mm. and 16 mm., respectively, in V_4). In both, the twelve lead tracings were normal in all other respects and hence were not included among the abnormals.

Abnormally deep and wide Q waves in aV_L or aV_F occurred in five of the fifteen abnormal tracings of the controls (33 per cent), in nine of the forty-eight abnormal tracings of angina patients without a history of infarction (19 per cent), and in fourteen of the twenty-seven with a history of infarction (52 per cent). Lead aV_L revealed this deep Q wave in one control subject, three Group I angina patients, and five Group II angina patients; this was accompanied by inversion of the T wave in aV_L in all but one patient with angina. In one control subject and one angina patient these were the sole abnormalities in the twelve leads. In four angina patients there was also inversion of the T wave in $V_{4,5,6}$. In the remaining two the T wave was inverted and there was a small Q in Lead I. In one of the latter there were deep Q waves in $V_{4,5,6}$ also. Lead aV_F showed abnormal Q waves in four control subjects, in six Group I angina patients, and in ten Group II angina patients. In one control subject and two of Group II angina patients, this was the sole abnormality in the twelve leads. In the remaining seventeen, the Q wave in aV_F was associated with inversion of T in Leads I or $V_{4,5,6}$, or both. In six subjects (two of each group) T in aV_F was also inverted.

Elevation of the RS-T segment, measuring 1 to 2.5 mm. in all leads except aV_R (where RS-T was depressed) occurred in one control subject and in no angina patients. Elevation of RS-T in $V_{1,2,3}$ only was found in two control subjects, in eight Group I angina patients, and in eight Group II angina patients. In all eighteen instances this was associated with other abnormalities, most often T-wave inversion. The degree of elevation ranged from 1.5 to 3 mm. in eleven instances and measured 1 mm. in the remaining seven.

Depression of RS-T in $V_{4,5,6}$ occurred in one control subject, in nine Group I angina patients, and in one Group II patient. Here again it was always associated with other abnormalities. The depression measured 1.5 to 3 mm. in four subjects and 1 mm. in the remaining seven.

Auricular flutter and auricular fibrillation each occurred once, both in angina patients. Left bundle branch block was found in one angina patient. Prolonged duration of P-R interval was found in two control subjects and two angina patients.

COMMENT

It is evident that the twelve-lead (like the three- or four-lead) electrocardiograms of patients with angina pectoris show no diagnostic characteristics. Only one-half of angina patients without a past history of myocardial infarction had abnormal tracings, and these abnormalities varied considerably. Patients with angina pectoris showed electrocardiographic abnormalities three to five times as frequently as did subjects without cardiac symptoms. The abnormalities were comparable in both groups, however, and it was impossible to tell from the tracings alone whether or not the individual was subject to attacks of cardiac pain.

The multiplicity of electrocardiographic patterns in patients with angina pectoris is not surprising in view of the multiplicity of pathologic findings in the coronary arteries and the myocardium of such patients.[10-12] As might be expected, abnormal tracings were encountered more frequently in patients with a past history of myocardial infarction. Fourteen out of thirty-four patients (41 per cent) showed abnormal Q waves in the unipolar limb leads suggestive of previous infarction; these fourteen comprised one-half of all the abnormal tracings in this group. In patients with a history of myocardial infarction, the electrocardiogram is not essential to indicate the presence of cardiac damage; nevertheless, seven out of thirty-four patients (21 per cent) had normal tracings.

When objective evidence of heart disease is not revealed by the physical examination or the electrocardiogram, the diagnosis of angina pectoris rests entirely on the clinical history. Master[13] has stated: "At least 40 per cent of patients with coronary disease do not present any objective evidence of heart disease. The physical examination, chest roentgenogram, and resting electrocardiogram all may appear to be entirely normal." Riseman and Brown[14] found that 26 per cent of patients with angina pectoris had no cardiac enlargement, no hypertension, and no abnormalities in the four-lead (I, II, III, IV F) electrocardiogram. In the present series, the occurrence of angina pectoris with normal twelve-lead electrocardiograms, normal heart size, normal blood pressure, and no history of past myocardial infarction was decidedly less frequent. Such patients constituted 13 per cent of the total 134 with angina and 17 per cent of the 100 angina patients without a past history of myocardial infarction (Table VII). On the other hand, only 29 per cent of subjects of comparable age but without cardiac symptoms fell into the same category.

The significance of the abnormal tracings found in 15 per cent of the present series of subjects without cardiac symptoms is not clear. There is ample evidence in the literature that abnormal electrocardiograms are not uncommon in apparently healthy subjects [4-6,8,9,15-17], the frequency of such findings varying with the criteria of normal, the age of the subjects, and the leads employed. Graybiel and his co-workers,[5] who studied Leads I, II, III, IV F, and IV R in

TABLE VII. EVIDENCE OF CARDIOVASCULAR ABNORMALITIES IN PATIENTS WITH ANGINA PECTORIS AND SUBJECTS OF SIMILAR AGE BUT WITHOUT CARDIAC SYMPTOMS

EVIDENCE OF ABNORMALITY	SUBJECTS WITHOUT CARDIAC SYMPTOMS (NO.)	PATIENTS WITH ANGINA PECTORIS (NO.)
Total Studied	100	134
Past history of myocardial infarction	0	34
Abnormal electrocardiograms*	15	48
Clinical cardiac enlargement*	0	21
Blood pressure of 165/100 mm. Hg or higher*	27	4
Blood pressure of 140/90 to 160/90 mm. Hg*	29	10
Subjects with none of the above characteristics	29	17

*In the remaining subjects.

1,000 healthy aviators 20 to 30 years of age, concluded: "The results serve to emphasize the great individual variation in the electrocardiographic pattern and the wide range of normal values . . . . it is apparent that the 'normal' extends well into what has commonly been regarded as the abnormal range." Taran and Kaye[17] found electrocardiographic (Leads I, II, III, and IV F) evidence of myocardial damage in 46.5 per cent of 102 subjects 60 to 90 years of age (ambulatory, not debilitated, not senile, and without any complaints which might be interpreted as cardiovascular in nature). They ask—"Are these changes a manifestation in the normal evolution of senescence of the cardiovascular system or the end result of a disease process?"

Re-evaluation of the clinical characteristics of the fifteen control subjects with abnormal tracings reveals the following. The age and sex distribution and the frequency of hypertension were similar to that of the entire group. All enjoyed normal or greater than normal physical activity and none had symptoms for many years. Their medical life histories were known through intimate personal contact and frequent periodic examination for 35 years in one, 20 to 28 years in four, 10 to 19 years in seven, and 1 to 2 years in the remaining three. In only two had major symptoms which might have affected the electrocardiogram been recognized. One, a man 64 years of age, had rheumatic fever with pericarditis thirty-two years previously. No evidence of residual cardiac damage had ever been discovered on physical examination and his blood pressure was 128/80 mm. Hg. His electrocardiogram showed deep Q waves in Leads III and aV_F with low amplitude T waves in Leads II and $V_{4,5,6}$. The other was a 72-year-old woman who had experienced recurrent paroxysms of auricular fibrillation for about eight years up to four years before this study. Her blood pressure was 142/70 mm. Hg. Her electrocardiogram showed diphasic T_I with inversion of T in $V_{5,6}$. Neither had shown any changes in their four-lead electrocardiograms during the past seven and twelve years respectively. Six other subjects in this group of fifteen had four-lead tracings during the preceding five to ten years. No changes were observed. The ages of these patients (two were 49, four were 50 to 59, five were 61 to 67, and four were 70 to 76 years of age) make it probable that many had coronary arteriosclerosis. It might be expected

that the prognosis of a group of subjects without cardiac symptoms but with abnormal electrocardiograms might be more unfavorable than for a similar group with normal tracings. This is not necessarily true in any individual case, however, and whether or not such subjects will develop cardiac symptoms before death is pure speculation.

A comparison of the abnormalities in the present series with those in acute myocardial infarction is of considerable interest. In the present series abnormal Q waves occurred in 17 per cent of patients with angina pectoris; elevation or depression of RS-T segments measuring 1.5 to 3 mm. were found in 11 per cent; and the combination of Q plus RS-T abnormalities occurred in only 2 per cent. These frequencies are much less than found in acute myocardial infarction. Serial electrocardiograms should reveal whether such abnormalities are constant (suggesting old damage) or progressive (indicating recent damage).

Time and experience have proved the value of the three standard leads and have also demonstrated their shortcomings. In theory, the unipolar limb leads should yield all the information which can be obtained from the bipolar standard leads. In practice, however, this is not true, not only because machines which record the three unipolar limb leads simultaneously are not available generally but also because the standards of normal, especially in regard to the T waves, are not comparable. Although the unipolar limb and precordial leads do much to overcome the shortcomings of the standard leads, considerably more experience must accumulate before the unipolar can replace the bipolar leads.

In the present series, the combination of bipolar standard, unipolar augmented limb leads, and unipolar multiple precordial leads together gave a more complete picture than was obtained from a study of any one of the three types of leads alone. In most instances the findings in the standard, precordial, and limb leads corroborated each other. In other instances the findings in the standard leads were modified or explained by the findings in limb or precordial leads. For example, the unipolar limb leads were of particular importance in determining the significance of Q waves in Leads I or III; similarly, the precordial leads served to separate right from left bundle branch block. In other instances unequivocal evidence of damage was found in one lead only; for example, deep Q waves, which may be evidence of old myocardial infarction; were often found in aV_F only.

SUMMARY AND CONCLUSIONS

1. A study of the twelve-lead electrocardiograms (three bipolar standard, three augmented unipolar limb, and six unipolar precordial leads) in 100 control subjects without symptoms or signs of heart disease and 134 patients with angina pectoris revealed no diagnostic characteristics.

2. Abnormal electrocardiograms were found in 15 per cent of the control subjects, 48 per cent of the angina patients without a past history of myocardial infarction, and 79 per cent of the angina patients with a past history of infarction. Although abnormal electrocardiograms were encountered more frequently in patients with angina pectoris than in those without cardiac symptoms, the ab-

normalities were comparable in both groups, and it was impossible to state from the tracings alone whether or not the person was subject to attacks of cardiac pain.

3. The abnormalities encountered most frequently were inverted, diphasic, or absent T waves in Leads I and $V_{5,6}$. Abnormal Q waves were less frequent and were most common in angina patients with a past history of myocardial infarction. Elevation or depression of RS-T segments was not infrequent and was usually, but not always, accompanied by abnormalities of Q or T. The frequency of abnormalities of Q, RS-T, and T in patients with angina pectoris and also in subjects without cardiac symptoms emphasizes the necessity of searching for progressive changes in serial electrocardiograms before making a diagnosis of acute myocardial damage.

4. Normal twelve-lead electrocardiograms, normal heart size on physical examination, and normal blood pressure occurred in 17 per cent of angina patients without a history of past infarction and in 29 per cent of subjects without cardiac symptoms. Such evidence is of little value, therefore, in separating patients with angina from those without cardiac symptoms.

5. The twelve-lead electrocardiograms gave more information of value than could be obtained from the three standard plus one apical lead. It is advisable to take twelve-lead tracings routinely in all cases and not only when cardiac damage is suspected.

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PULMONARY STENOSIS WITH PATENT FORAMEN OVALE

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IN THIS communication there is reported a study of a patient with pulmonary stenosis and patent foramen ovale which revealed two hitherto unreported diagnostic features believed to be of importance. In addition, the change in blood oxygen content observed during partial occlusion of the stenotic pulmonary valve by a catheter permitted estimation of the size of the valve, the intracardiac right-to-left shunt, and the extracardiac collateral circulation to the lungs.

CASE REPORT

B. L., a Negro man, was first seen at Charity Hospital on Dec. 11, 1945, at the age of 22 years, when he entered the outpatient clinic with a chief complaint of dull, aching, epigastric pain of four hours duration. When he was very young his parents had been told that he had a leaking heart. He had always experienced dyspnea on exertion; as a boy, he could ride a bicycle six blocks and then had to stop to rest. He never squatted for relief. At about the age of 18 years he found he was unable to do heavy work. Since then his exertional tolerance had decreased. He had had frequent colds since childhood.

Physical examination revealed a blood pressure of 140/98 mm. Hg in the right arm, 135/95 mm. Hg in the left arm, and 155/110 mm. Hg in the left leg. There was moderate cyanosis of the lips and fingernail beds with clubbing of the fingers. The lungs were clear to percussion and auscultation. Generalized cardiac enlargement was present. There was a systolic thrill at the pulmonic area. The second sound was diminished at the base, particularly at the left of the sternum. A loud systolic murmur was heard over the entire precordium and back with point of maximal intensity in the second left intercostal space. A moderately loud, high-pitched diastolic murmur was heard best in the same location and was transmitted toward the apex. The liver edge was palpated 3 cm. below the right costal margin. No edema was present.

The red blood cell count was 6.83 million per cubic millimeter; hemoglobin was 19.5 Gm. per 100 c.c.; the packed red cell volume was 62 per cent. Serum protein was 6.4 Gm. per 100 c.c. One plus albuminuria was present. Plasma volume by the Evans blue method was 2,500 c.c.; total blood volume was calculated to be 6,570 c.c.

The electrocardiogram showed high, wide, notched P waves, wide QRS complexes, and marked right axis deviation (Fig. 1). It was compatible with marked right ventricular hypertrophy and atrial enlargement.

Roentgenologic examination showed abnormally clear lung fields, concavity of the pulmonary arc of the left cardiac border, a prominent, nonpulsating pulmonary artery below the aortic knob, and enlargement of the right ventricle (Fig. 2). Angiocardiograms revealed delayed emptying time of the right ventricle with contrast medium still in the right ventricle and pulmonary artery in the twelve-second plate. Dilatation of the pulmonary artery was demonstrated. At each interval following injection of the dye the pulmonary artery was better visualized than the aorta (Fig. 3).

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During the next two and one-half years the patient had five admissions to the hospital and was seen at weekly intervals in the cardiac clinic.

Respiratory function tests were performed on May 21, 1948, with a Benedict-Roth apparatus utilizing 100 per cent oxygen. At rest his ventilation during a four and one-half-minute test was 49,320 c.c. per minute, and he absorbed 285 c.c. of oxygen per minute. While exercising by moving his legs against resistance during a three-minute test his ventilation was 69,070 c.c. per minute, and he absorbed 395 c.c. of oxygen per minute. While at rest he thus absorbed 5.8 c.c. of oxygen per liter of ventilation; during exercise the ratio was 5.7 c.c. of oxygen per liter of ventilation.

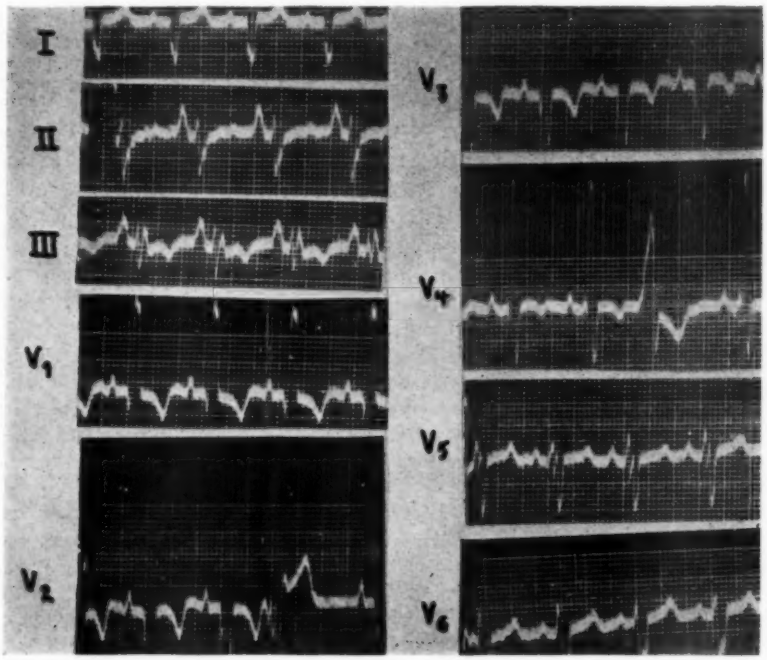


Fig. 1.—Electrocardiogram, August, 1948. The tracings show wide QRS complexes with prominent R_{V_1} and S_{V_6}. This picture is compatible with marked right ventricular hypertrophy. The Q_{V_1} suggests that right bundle branch block may not be present.

Twice during the terminal months of his illness arterial puncture and cardiac catheterization were carried out (Tables I and II). The arterial oxygen saturation at rest was 64 per cent on May 18, 1948, and 51 per cent six months later. During the first catheterization the tip of the catheter was passed with difficulty into the pulmonary artery. When passage to this point had been effected, the patient became extremely dyspneic and apprehensive, and it was deemed wise to terminate the procedure at once. The pulmonary artery was catheterized without untoward incident at the later date. Values for blood oxygen obtained at catheterization reflect the arterial anoxemia and reveal a wide arteriovenous difference. Low pressure in the pulmonary artery and extremely high right ventricular systolic pressure indicated stenosis of the pulmonary valve (Fig. 4). Myocardial failure was shown by the high right ventricular diastolic and right atrial pressures. There was no evidence of left-to-right shunt.

Dyspnea became progressively worse, cyanosis increased, and exercise tolerance was reduced to the point at which the patient required absolute bed rest. There were repeated acute episodes of accentuation of dyspnea and cyanosis accompanied by a shocklike state which would persist for one to three hours and was partially relieved by inhalation of 100 per cent oxygen. Therapy consisted of digitalization, salt-free diet, and limitation of activity.

TABLE I. OXYGEN STUDIES ON ARTERIAL BLOOD

DATE	INSPIRED GAS	ACTIVITY OF SUBJECT	O_2 CONTENT (VOL. %)	O_2 CAPACITY (VOL. %)	O_2 PER CENT SATURATION	A-V DIFFERENCE (VOL. %)	α*	I†
May 18, 1948	Air	Resting	17.9	7.82	64	5.6‡	0.60	0.06
	Oxygen	Resting	20.5	27.8	73			
	Oxygen	Exercising§	16.1	27.3	59			
Nov. 8, 1948	Air	Resting	15.2	29.8	51	5.7‖	0.68	0.08
	Oxygen	Resting	18.5	29.8	62			

*Net right-to-left shunt, calculated as described elsewhere.[1]

†Oxygen unsaturation of blood that has flowed through pulmonary capillaries in relation to functioning alveoli, calculated as described elsewhere.[1]

‡Difference between right atrial oxygen content on May 5, 1948, and arterial content on May 18, 1948, the latter adjusted to correspond with the oxygen capacity of the right atrial sample.

§Sample withdrawn immediately following twenty deep knee bends.

‖Difference between right ventricular oxygen content on Oct. 28, 1948, and arterial content of Nov. 8, 1948; oxygen capacities of the two samples are the same.

TABLE II. RESULTS OF VENOUS CATHETERIZATION. SUBJECT AT REST, BREATHING AIR

DATE	SOURCE OF SAMPLE	O_2 CAPACITY (VOL. %)	O_2 CONTENT (VOL. %)		PRESSURE† (mm. Hg)
			OBSERVED	ADJUSTED*	
May 5, 1948	Superior vena cava	27.6	13.3	13.4	—
	Inferior vena cava	27.1	13.5	13.9	—
	Right atrium	28.7	12.8	12.4	—
Oct. 28, 1948	Right pulmonary artery	29.8	5.9	—	9/5
	Right ventricle	29.8	9.5	—	177/9
	Right atrium	—	—	—	25/0
	Superior vena cava	29.8	9.7	—	—

*Oxygen content adjusted to correspond to average oxygen capacity of the group.

†Systolic over diastolic pressure estimated with Statham strain gauge manometer, and calculated from a point 13 cm. above the back, patient supine. There is considerable artifact in the tracing taken from the pulmonary artery (Fig. 4), and the estimated pressure therefrom is only approximate.

A diagnosis of the tetralogy of Fallot was made. It was decided to perform a Blalock type of operation; however, the patient pursued a rapidly downhill course and died on Nov. 18, 1948, before any surgical procedure was attempted.

Post-mortem examination (performed by Dr. H. W. May): The heart weighed 560 grams. The right ventricular wall was 2.5 cm. in thickness, and there were marked right ventricular and atrial hypertrophy and dilatation. The left ventricular wall measured 1.5 cm. and hypertrophy of this chamber was minimal. There was fusion of the pulmonary valve cusps resulting in a diaphragm with a 2.5 mm. central opening (Fig. 5). The foramen ovale was closed by a thick membrane which was unattached anteriorly and overlapped the edge of the foramen in such a way as to permit the tip of the little finger to pass between the two atria (Fig. 5). Circumference of the tricuspid valve was 12.5 cm.; of the pulmonary valve, 4 cm.; of the mitral valve, 10.5 cm.; and of the aortic valve, 6 cm. There were well-developed collateral vessels throughout the chest

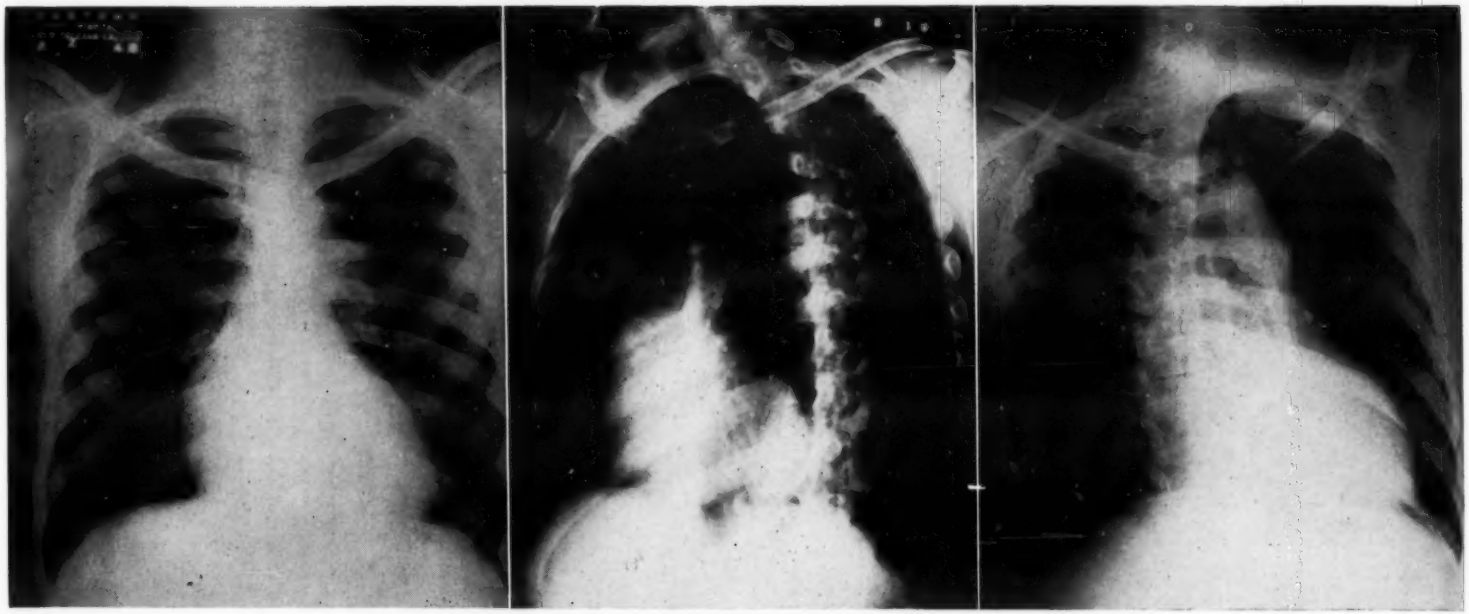


Fig. 2.—Posteroanterior, left anterior oblique, and right anterior oblique x-rays, August, 1948. Position of patient for the oblique views is not good. At fluoroscopy in the left oblique position right ventricular enlargement was evident, the left ventricle cleared the spine normally, and the dilated pulmonary artery was readily demonstrable.

Fig. 3.—Angiocardiograms 3, 5, 9, and 12 seconds after injection of 45 c.c. of 70 per cent Diodrast.* January, 1946. Left anterior oblique position. Emptying time of the right ventricle is greatly prolonged, and complete filling of the dilated pulmonary artery is delayed. The aorta is visualized at each interval but is less well filled than the pulmonary artery.

*Iopyracyl (Diiodopyridone acetic acid diethanolamine—Winthrop Chemical Company, Inc., New York, N. Y.).

cavity. The lungs were acutely congested. The liver weighed 1,340 grams and presented no abnormalities to gross inspection.

Microscopic study revealed the following pertinent features: The myocardium was extremely vascularized, and fibers of the ventricles were hypertrophied. The coronary arteries showed no abnormalities. The renal parenchyma contained numerous hyalinized glomeruli surrounded by atrophic tubules; Bowman's capsule showed some hyalinization, more marked in the small glomeruli. There were no abnormalities of the lungs or liver.

CIRCULATORY HEMODYNAMICS

From the data assembled in Table I the values in the last two columns have been calculated in a manner described elsewhere.[1] The net right-to-left shunt, α, is that amount of mixed venous blood which reaches the systemic arterial circulation without having passed through the lungs. The figures indicate the shunt to have been 60 per cent on May 18, 1948, and 68 per cent six months later. The oxygen unsaturation of blood which has flowed through capillaries in the walls of functioning alveoli, l, was normal on each occasion. The figures, 0.06 and 0.08, indicate that the oxygen saturation of pulmonary venous blood was 94 per cent and 92 per cent, respectively. The arterial anoxemia of this patient was thus entirely attributable to the shunting of venous blood through the foramen ovale from the right into the left atrium.

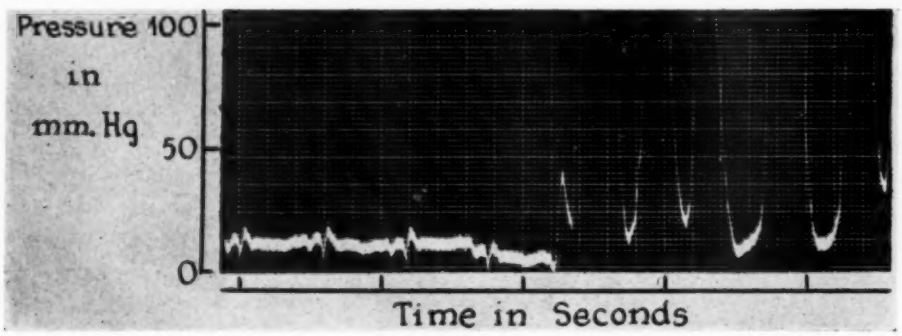
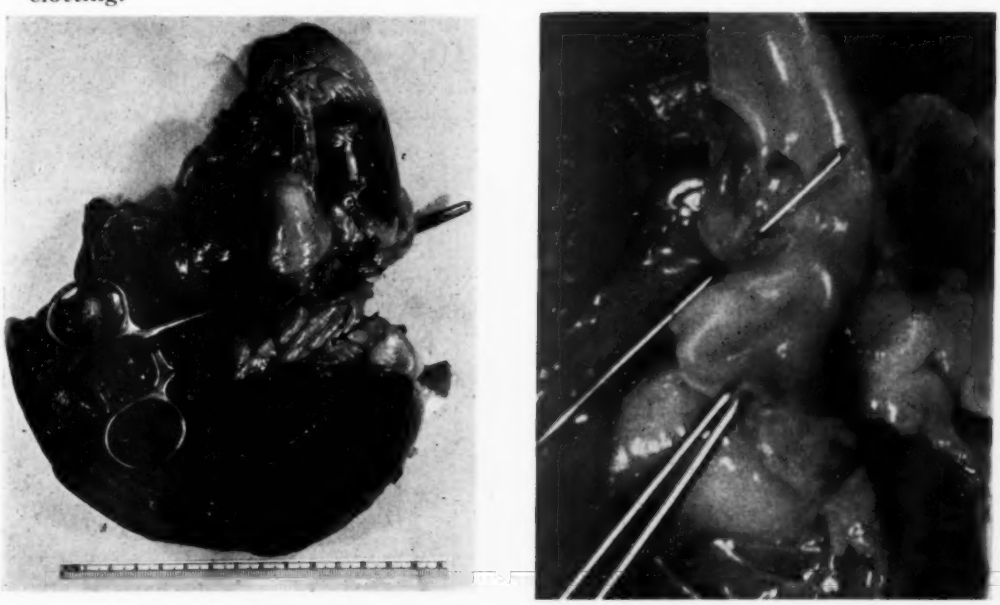


Fig. 4.—Continuous pressure recording during retrograde passage of the catheter tip from the pulmonary artery into the right ventricle. Tracing reads from left to right. The patient is supine. The pressure scale is uncorrected. "O" pressure corresponding to the level of the patient's back. Pressures are read from the top of the string shadow. There is considerable artifact in the pulmonary artery tracing and some overshooting of the ventricular diastolic troughs. The uncorrected ventricular systolic pressure, not shown here, is seen in another segment of the tracing with a less sensitive manometer to be 187 mm. of mercury.

The lower oxygen content of pulmonary arterial blood observed on Oct. 28, 1948 (Table II), in comparison with that in the right ventricle and superior vena cava, is believed to be of considerable significance. It is thought to have been brought about by partial occlusion of the stenotic pulmonary valve by the catheter with consequent increase in the magnitude of the right-to-left shunt due to passage through the foramen ovale of blood to which passage through the valve was denied. The increased admixture of venous with arterial blood brings about a reduction in the oxygen content of the latter and this, in turn, as long as

the arteriovenous oxygen difference remains unchanged, effects a lower venous oxygen content. The progressive fall in arterial and venous oxygen continues until a new equilibrium is established at a lower level of content. Many minutes are necessary for the achievement of such an equilibrium, and it may be that the oxygen content of the pulmonary arterial blood would have been even lower had more than a few minutes elapsed following introduction of the catheter through the stenotic valve. Recovery to the initial state of equilibrium is believed to have taken place in the ten-minute period that elapsed between the removal of samples from pulmonary artery and right ventricle, since the oxygen content of the latter agrees well with the content of superior vena caval blood removed some minutes later. Samples were taken from other locations but not analyzed because of clotting.



A. B.

Fig. 5.—Heart. *A*, Clamp passes through the foramen ovale from right to left. The stenosed pulmonary valve is seen from the arterial side (arrow). Note the greatly thickened right ventricular wall. *B*, Probe passes from right ventricle to pulmonary artery through the stenosed pulmonary valve.

The mechanism of lowering of the oxygen content of the pulmonary arterial blood postulated here could be tested in several ways:

1. There might be a change in the character of the murmur during the presence of the catheter in the valve;

2. A sample taken immediately after the tip has entered the pulmonary artery should show the same oxygen content as right ventricular blood, then progressively fall for several minutes;

3. When the oxygen content of pulmonary arterial blood has stabilized at a lower level, the content of oxygen in systemic arterial blood should have fallen by an amount equal to the difference between the right ventricular and pulmonary arterial oxygen content.

TABLE III. CALCULATED BLOOD FLOW THROUGH VARIOUS CHANNELS, EXPRESSED AS FRACTIONS OF THE SYSTEMIC BLOOD FLOW

ASSUMED PRECAPILLARY LEFT-TO-RIGHT SHUNT IN LUNGS (γ)	CORRESPONDING DIAMETER OF PULMONARY VALVE ORIFICE	SYSTEMIC BLOOD FLOW (1.0)	LEFT VENTRICLE OUTPUT ($1 + \gamma$)	RIGHT-TO-LEFT SHUNT		FLOW IN PULMONARY CIRCULATION		
				ACROSS ATRIAL DEFECT (α')	NET (α)	PULMONARY ARTERY FLOW ($1 - \alpha'$)	PULMONARY CAPILLARY FLOW ($1 - \alpha' + \gamma$)	EFFECTIVE PULMONARY FLOW ($1 - \alpha$)
Minimal – 0	6.25 mm.	1.0	1.0	0.68	0.68_3	0.32	0.32	0.32
Maximal – 0.36	2.5 mm.		1.36	0.93		0.07	0.43	

None of these tests were attempted in this patient.

From the fall in pulmonary arterial oxygen content produced by the passage of the catheter through the pulmonary valve, it is possible to calculate blood flow through the valve, through the foramen ovale, and through precapillary anastomoses between bronchial and pulmonary arteries. Assumptions are made that a state of equilibrium was attained before the pulmonary arterial sample was removed, and that the only alterations in circulatory dynamics were a decreased flow through the obstructed valve and an increased right-to-left shunt of equal magnitude through the foramen ovale. There is further assumed to have been a fall in arterial oxygen content of 3.6 volumes per 100 c.c., equal to the difference between right ventricular and pulmonary arterial content, so that the arterial oxygen saturation would have been 39 per cent when the catheter was in the pulmonary valve. The net right-to-left shunt before passage of the catheter through the pulmonary valve, 68.3 per cent (Table III), would, during the presence of the catheter in the valve, rise to 73.6 per cent.

All of the factors are now known which permit one to define the possible extremes of flow through the channels indicated above. It is assumed that any precapillary left-to-right shunt in the lungs remains constant. If the catheter completely occludes the stenotic pulmonary valve, the intracardiac right-to-left shunt becomes equal to the systemic blood flow, and the maximal possible value for extracardiac left-to-right shunt may be calculated. If, on the other hand, there is no extracardiac left-to-right shunt, the reduction in pulmonary flow brought about by the presence of the catheter in the pulmonary valve is due entirely to the encroachment of the catheter on the cross section of the blood stream at the valve. The relationships between these variables have been elucidated in another paper[2] and are contained in the expression,

$$\alpha = \frac{\alpha'}{1 + \gamma}$$

in which α is the net right-to-left shunt, α' is the intracardiac right-to-left shunt, and γ is the extracardiac left-to-right shunt. All symbols refer to fractions of the systemic blood flow, the latter being expressed as unity (1.0).

If the first extreme in the foregoing paragraph is postulated, during catheterization of the pulmonary artery α' becomes equal to the systemic blood flow, 1.0, and α, as already indicated, equal to 0.736. By substitution in the above equation,

$$0.736 = \frac{1.0}{1 + \gamma} \qquad \text{On solution, } \gamma = 0.36.$$

This is the maximum possible extracardiac shunt from left to right—36 per cent of the systemic blood flow. The output of the left ventricle would be equal to $1 + \gamma$ or 1.36 times the systemic blood flow; the fraction of the left ventricular output shunted to the lungs is $\frac{\gamma}{1 + \gamma}$* or 0.26. If the extracardiac left-to-right

*This is the β of Lundsgaard and Van Slyke.[3]

shunt remains constant at 36 per cent of the systemic blood flow, when the catheter is removed and the net right-to-left shunt reverts to 0.683, the intracardiac shunt can be calculated by substituting 0.683 for α and 0.36 for γ in the equation:

$$0.683 = \frac{\alpha'}{1 + 0.36} \qquad \text{On solution, } \alpha' = 0.93.$$

The flow through the pulmonary valve is then $1 - \alpha'$ or 0.07 times the systemic blood flow. The total amount of blood entering the pulmonary veins through capillaries in relation to functioning alveoli—the pulmonary capillary flow of Bing, Vandam, and Gray[1]—is then equal to $1 - \alpha' + \gamma$ or 0.43 times the systemic blood flow.

If complete absence of any extracardiac left-to-right shunt is assumed, the intracardiac right-to-left shunt becomes identical with the net right-to-left shunt. The increase in shunt brought about by passage of the catheter through the valve is 0.736 − 0.683 or 0.053. The unobstructed flow through the valve is one minus the shunt (1.0 − 0.683) or 0.317. Since the decrement in flow through the valve when the catheter is passed is equal to the increased shunt, the flow through the valve is reduced by 0.053/0.317 or approximately one-sixth. Thus, the cross sectional area of the catheter must be approximately one-sixth that of the stenotic valve orifice. This corresponds to a valve diameter of approximately two and one-half times that of the catheter, or 6.25 mm.

The maximal and minimal fractions of blood that would pass through various channels under the extreme circumstances indicated above are assembled in Table III. The true values lie somewhere between these extremes, but for numerous reasons it is believed that the situation of maximal extracardiac left-to-right shunt and complete occlusion of the pulmonary valve by the catheter was closely approximated in this patient.

1. It is known that extensive extracardiac collateral circulation develops in cyanotic congenital heart disease, the extent of the collateral circulation varying directly with the severity of the arterial anoxemia and the age of the subject.[5,6]

2. Such collaterals were observed grossly at post-mortem examination.

3. The diameter of the 8 Fr. catheter, 2.5 mm., is the same as the diameter of the valve orifice as measured at post-mortem examination, at which time the catheter was demonstrated to fit snugly in the opening; it is unlikely that the opening would have allowed the passage of much blood around the catheter during life. Certainly it could not have dilated to 6.25 mm. in diameter, the size postulated from the calculations above.

4. The maximal left ventricular output, 1.36 times the systemic blood flow, would not be attended with great increase in the size of the left ventricular cavity and is consistent with the "minimal hypertrophy" of the left ventricle described at post-mortem examination.

The actual volumes of blood flowing through the various channels can be calculated by multiplying the systemic blood flow by the appropriate factors.

The systemic blood flow, calculated according to the Fick principle from figures for arterial and venous oxygen and oxygen uptake observed on different days,

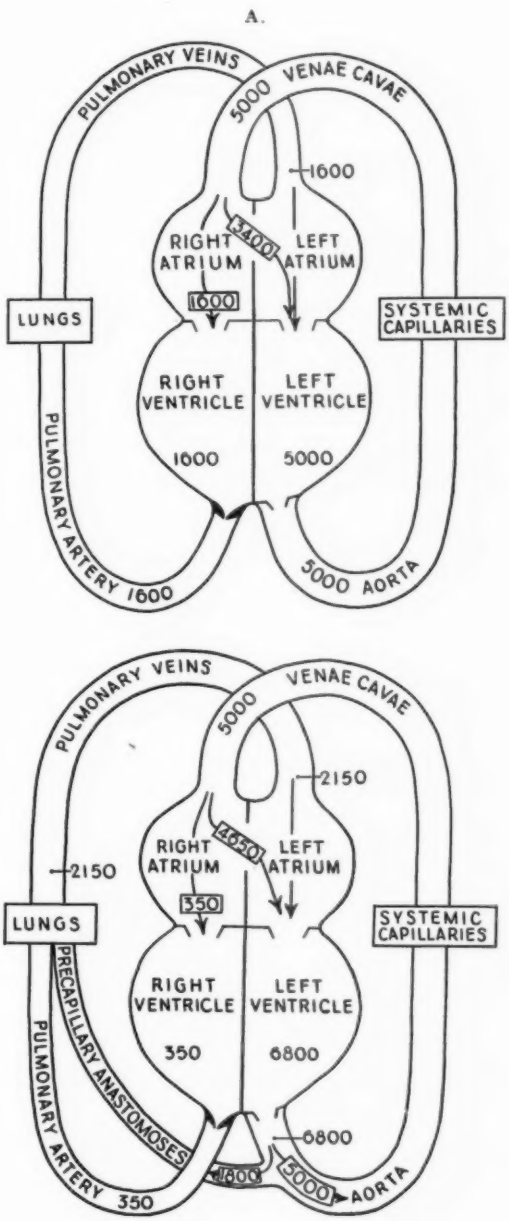


Fig. 6.—Calculated volumes of blood flow per minute through various channels. In each situation the net right-to-left shunt is 3,400 c.c. per minute, the effective pulmonary flow 1,600 c.c. *A*, No collateral flow to the lungs; estimated diameter of pulmonic valve orifice 6.25 mm. *B*, Maximum possible pulmonary precapillary anastomoses; estimated diameter of pulmonic valve orifice 2.5 mm.

is 5,000 c.c. per minute. This figure, multiplied by the various factors of Table III, yields the results depicted schematically in Fig. 6.

DISCUSSION

The three features essential in arriving at the decision to do a Blalock type of operation were all present in this patient. They are here enumerated together with confirmatory observations.

1. A right-to-left shunt must be present. The severe arterial anoxemia represented, as indicated in the foregoing section, a net right-to-left shunt of 60 per cent of the systemic venous return to the heart on May 18, 1948, and 68 per cent six months later.

2. Blood flow through the lungs must be diminished. This is suggested by the diminution of normal vascular markings in the x-ray, by the failure during fluoroscopy to observe pulsations in the dilated pulmonary artery, and by the lack of rise in oxygen consumption per liter of air ventilated during muscular exercise.[5] Reduced pulmonary blood flow is usually associated with stenosis or atresia of the pulmonary valve or infundibulum of the right ventricle. Pulmonary stenosis was suggested by the quality of the second heart sound on physical examination and by the delay in passage of contrast medium from the dilated right ventricle into the dilated pulmonary artery (Fig. 3). Stenosis was confirmed by the finding of low pressure in the pulmonary artery in contrast to the high right ventricular pressure (Fig. 4). The maximum possible flow to the lungs, as calculated in the foregoing section, was less than half the systemic blood flow (Table III, Fig. 6).

3. Significant impairment of the individual's activity must be present. The history as recorded attests that this criterion was met. Objective evidence was found in the polycythemia and clubbing, the marked overventilation and low ratio of oxygen absorbed to air ventilated, the severe and progressive arterial anoxemia, the prompt fall in arterial oxygen saturation following twenty deep knee bends (Table I), the elevated right ventricular diastolic and right atrial pressure (Table II), and the arteriovenous oxygen differences of 5.6 and 5.7 volumes per 100 c.c. (Table I), which are considerably higher than would be expected in a person with arterial unsaturation of this degree.[1]

Proper evaluation of two features could have led to the correct ante-mortem diagnosis. The long delay of the contrast medium in the dilated right ventricle and the concomitant poor visualization of the aorta suggest that ventricular septal defect with an overriding aorta was not present. Further evidence that the right ventricle was not in direct communication with the aorta is to be found in the disparity between right ventricular and systemic systolic pressures. These values would be expected to approximate each other closely in the tetralogy of Fallot. The right ventricular systolic pressure on Oct. 28, 1948, was 35 to 75 mm. of mercury higher than that determined in the brachial artery at various times with the sphygmomanometer, and 59 mm. higher than the femoral arterial systolic pressure of 118 mm. of mercury recorded with a strain gauge manometer on Nov. 8, 1948. The demonstrated coexistence of pulmonary stenosis and a

right-to-left shunt, together with the probable absence of a ventricular septal defect, could only point to the presence of a defect of the atrial septum.

Although but twenty-nine cases of pulmonary stenosis with atrial septal defect have hitherto been reported,[7] it is not unlikely that the condition is more prevalent than recognized.* In Abbott's series there are sixteen instances of this anomaly, eighty-five of the tetralogy of Fallot, and thirty of pulmonary atresia with dextraposition of the aorta and ventricular septal defect.[8] This is an incidence of 12 per cent among the anomalies with which it is most likely to be confused. It is likely that many cases considered to be the tetralogy of Fallot and for which a Blalock type of operation has been carried out are in reality instances of pulmonary stenosis with atrial septal defect. In nine of seventeen cases of the tetralogy of Fallot in which comparative data are available, reported by Bing, Vandam, and Gray,[5] the disparity between right ventricular and femoral arterial systolic pressures was 29 mm. of mercury or greater. The pressures were not simultaneously recorded, and the divergence in many instances is undoubtedly attributable to the difference in time of observation. In three cases this can hardly be the explanation, since the differences in pressure were 70, 81, and 96 mm. of mercury. It is suggested that two of these three cases, at least, may be examples of pulmonary stenosis with atrial septal defect. (In the third case the right ventricular pressure was normal, and one is inclined to question the diagnosis of pulmonary stenosis.) In seven of the nine cases, including the three just cited, the right ventricular systolic pressure was lower than the femoral arterial. Since the right ventricular pressure in patients with an intact ventricular septum is independent of the systemic pressure, it is apparent on theoretical grounds that the right ventricular systolic pressure could be higher than, the same as, or lower than the systemic systolic when pulmonary stenosis is present.

Although the case here reported presented only an anatomic patency of the foramen ovale, from a functional standpoint there was a defect of the atrial septum which permitted blood to flow from right to left. It is inferred from the patient's clinical progress and borne out by the progressive anoxemia demonstrated by the two analyses of arterial blood that the right-to-left flow through the foramen ovale increased as the myocardium failed. From analogy with uncomplicated atrial septal defect, in which the flow is from left to right, it is clear that obstruction to the normal outflow of blood from the right atrium, with consequent elevation of the pressure in that chamber, must be present in any case of atrial septal defect before blood can flow from right to left. In the syndrome here discussed the obstruction is brought about by failure of the right ventricle.

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*To the cases published elsewhere and the one here reported, we add that of a 6-year-old Negro child who was admitted to Charity Hospital on Jan. 9, 1949, and expired twenty-eight hours later with a brain abscess and purulent meningitis. Clinically she presented cyanosis, clubbing, bulging precordium, a loud systolic heart murmur, and absence of hepatic enlargement. Roentgenographic and electrocardiographic studies were not carried out. There was a 2 mm. opening in the center of a diaphragm-like pulmonic valve and a small functional patency of the foramen ovale.

SUMMARY

1. A case of pulmonary valvular stenosis with patency of the foramen ovale is reported.

2. Prolonged retention of the contrast medium in the right ventricle on angiocardiography and marked disparity between the right ventricular and systemic arterial systolic pressures were observed. These signs in a subject with a right-to-left shunt and pulmonic stenosis are presumably pathognomonic of an intact ventricular septum and patent atrial septum.

3. A fall in blood oxygen content, detected when the catheter was passed into the pulmonary artery, is believed to represent an increase in the right-to-left shunt consequent to obturation of the stenosed pulmonary valve by the catheter. From the magnitude of the change in blood oxygen content, the probable flow of blood through the atrial defect, through the pulmonary valve, and through extracardiac precapillary collaterals to the lungs has been calculated.

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CONGENITAL AORTIC SEPTAL DEFECT

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A CONGENITAL aortic septal defect is defined as an opening in the contiguous walls of the pulmonary artery and the aorta above the valves without signs of inflammation or aneurysm, the defect being due to incomplete division of the truncoconal channel by the truncoconal septum. Such a defect has been reported infrequently. Thirteen cases appear in the literature.[1-13] Several others considered as such must be rejected because of uncertainty as to the congenital nature of the opening or for other reasons. The case of Oberwinter[14] and that of Moorhead and Smith[15] quite possibly were examples of rupture of the septum in later life. Cazin's[16] patient was not autopsied, and the diagnosis was based on clinical findings alone. The defect in Lutembacher's[17] patient was actually an abnormally short ductus arteriosus, the pulmonary artery and aorta approximating. The patient of Shepherd, Park, and Kitchell[18] cannot be included because an aneurysm was present into which both the aorta and the pulmonary artery opened so that the connection was not direct. In addition to the thirteen cases mentioned above, it should be noted that Potts[19] encountered four cases, and Dexter[20] demonstrated such a defect during cardiac catheterization when the catheter passed from the pulmonary artery into the aorta. Details of these cases have not yet been published.

CASE REPORT

The following report is particularly interesting in that thoracic aortography gave striking evidence of the defect.

N. M., a 9-year-old white child, was referred to Hahnemann Hospital for evaluation of her cardiac condition. Delivery had been at term after a normal pregnancy. The family history was remarkable only in that a sibling brother who died at the age of 4 days was said to have been a "blue baby." No details as to this infant were available. On discharge from the hospital the mother was told that this patient had a patent ductus arteriosus. At home her respirations were noisy and difficult. Her panting could be heard at a great distance from her crib. There was no cyanosis and feedings were taken without distress. At the age of 5 weeks she suddenly became limp, turned blue about the mouth, and gasped for breath. The episode lasted about five minutes and there appeared to be no residua. By the age of 3 months there had been improvement in her respirations; they were no longer noisy or difficult. Thereafter for several months there were no apparent abnormal signs except for very forceful cardiac action and discomfort when lying in the prone position. At the age of 18 months she was studied at another clinic, and a diagnosis of patent ductus arteriosus was made. Operation was advised but as there was some question as to the certainty of the diagnosis the parents would not consent. Until the age of 2 years there were no untoward happenings. Then she began to have frequent episodes of epistaxis which continued periodically to the time of admission to this hospital. At the age

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of 3 years she complained one day of feeling unwell. The following morning she was found in bed unconscious and very cyanotic about the mouth. After five minutes her normal color had returned and she was fully conscious, appearing to have suffered no ill effects. One year later there was a similar episode. Again there was transient cyanosis and unconsciousness with complete recovery after a short time. During her fifth year she developed pneumonia and was hospitalized. During the course of this illness she complained one day of very severe precordial pain, radiating down her left arm. Morphine was resorted to as the attacks occurred several times during the course of the day. Unfortunately, the hospital records were not available. At the age of 8 years she again had "pneumonia." Cardiac pain recurred during this illness and again morphine was required for its relief. There was evidently some liver involvement at this time although the mother did not know its nature. Following this illness and a prolonged convalescence the child's health was fairly good. She had frequent epistaxis and sometimes complained of headache and dizziness, but her activities were not limited. Twelve days before admission to this hospital, while sleeping with her mother, cardiac action became so forceful that the bed vibrated. She complained of severe pain in her chest and seemed to have difficulty in breathing. Morphine seemed to give relief, and she was comfortable for the next several hours. However, the following night she again complained of pain, thoracic and abdominal, and hospitalization was advised. She was admitted to another hospital where it was recorded that her cardiac rhythm was very irregular and that her liver was tender and extended 6 cm. below the costal margin. She was treated with penicillin, phenobarbital, digitalis, and oxygen and after some degree of improvement was transferred to this hospital.

On admission the child was found to be poorly developed and nourished, her height and weight being low normal for her age. She was somewhat pallid, but there was no cyanosis. Pertinent physical findings were confined to the cardiovascular system. There were visible strong pulsations in the vessels of the neck. The bony thorax was asymmetrical, there being a pronounced precordial bulge. The cardiac impulse was extremely forceful and irregular and was visible over the entire anterior chest and upper abdomen. Its maximum intensity was in the seventh interspace outside the anterior axillary line. There were no thrills palpable. The sounds were of fair quality. There was no accentuation of the pulmonic second sound. The rhythm was grossly irregular. The apical rate was 110 per minute. At the base of the heart to the left of the sternum was heard a Grade III systolic murmur; there was also an early blowing diastolic murmur. In the mitral area were heard a Grade III systolic and a later diastolic murmur; the latter was somewhat rumbling in character and of a lower pitch than that at the base. A smooth, nontender liver edge was palpable 1.5 cm. below the costal margin. There was no clubbing or cyanosis of fingers and toes. All peripheral pulses were readily palpable. The blood pressure in the upper extremities was 104/60 mm. Hg and in the lower extremities 129/60 mm. Hg.

Laboratory data: The erythrocytes ranged between 3.1 and 4.2 million, the hemoglobin between 9.6 and 12.9 Gm. The leucocytes numbered in the neighborhood of 9,000 on several occasions with a slight preponderance of polymorphonuclears. The sedimentation rate was normal. Several blood cultures were negative. On occasion the urine contained a trace of protein but otherwise was normal. An electrocardiogram (Fig. 1) on admission showed auricular fibrillation with a very marked right axis deviation. The RST segment was depressed in Leads II and III; the T waves were flat in Lead I. Subsequent studies on several occasions showed no change. Fluoroscopy, chest films, and esophageal studies showed the peripheral lung fields to be clear. There was marked dilatation of the pulmonary vessels and a moderately intense hilar dance. The pulmonary artery was considerably dilated. The heart was markedly enlarged on frontal silhouette and also in depth. The esophagus was displaced posteriorly, particularly in the region of the left auricle, and was somewhat compressed. The pulmonary conus of the right ventricle was definitely enlarged (Fig. 2).

On admission a diagnosis of congenital heart disease with possible rheumatic mitral valvular disease was made. Before definitive studies were considered an attempt was made to improve her compensation. She was digitalized and maintained on bed rest for one month. There was great improvement in her general condition, but the fibrillation persisted. Because the possibility of an interauricular septal defect with a concomitant mitral stenosis was entertained, cardiac

catheterization was performed when her improvement had persisted for some time. Because of the existing fibrillation it was deemed unwise to enter the right ventricle and invite even transient further disturbances of rhythm. Therefore, pressures and blood samples were obtained only from the superior and inferior venae cavae and from the right auricle. No left-to-right shunt was demonstrated. There were no ill effects from the procedure. After further clinical observation it was decided to perform thoracic aortography in the hope of demonstrating a patent ductus arteriosus or an aortic septal defect. The left axillary artery was exposed and opened, and a special radiopaque catheter inserted and guided under fluoroscopic control so that the open tip ultimately lay at the base of the ascending aorta just above the valves. Fifteen c.c. of 70 per cent Diodrast were injected speedily, the child being in an exaggerated right posterior oblique position.

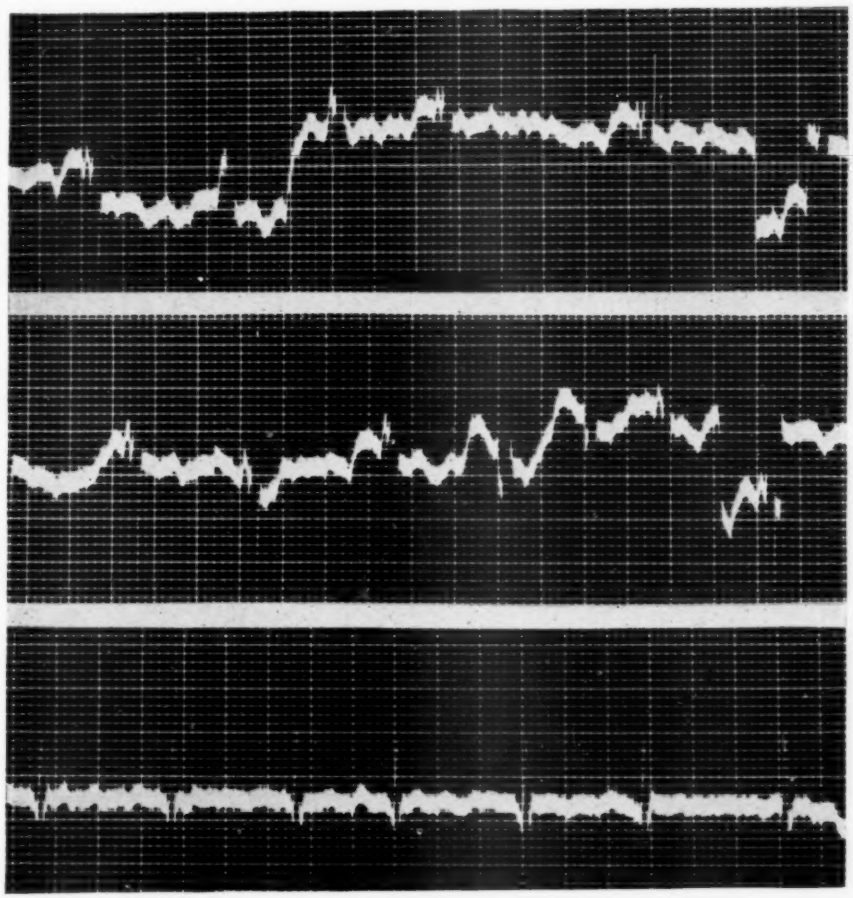

Fig.1.

There were no untoward effects from the procedure. Only one exposure was made, and when the film was viewed only a small amount of dye was seen in the ascending aorta; the arch and the descending portion were not visualized. However, the lesser circulation contained a goodly amount of dye (Fig. 3), indicating that it had been transferred directly from the aorta. No definite decision could be made as to whether this was due to a patent ductus arteriosus or an aortic septal defect. Exploration of the chest was decided upon after conference with the parents. It was reasoned that if she did have a patent ductus she would be greatly benefited by division and that the obvious risk of operation was justified. The initial stages of the operation were well tolerated.

Very shortly after the chest was opened it was noted that the cardiac action was satisfactory, the fibrillation having given way to a normal rhythm. However, when dissection of the great vessels was being carried out, the heart suddenly stopped. Intermittent cardiac compression was instituted immediately. There was an initial response, a slow, irregular rhythm being established. However, ventricular fibrillation developed which was stopped immediately with electric shock. A slow, normal rhythm was then re-established for a few moments, but thereafter there was no response despite prolonged massage.

Post-mortem examination showed the heart to be greatly enlarged, weighing 350 grams. The right ventricle was hypertrophied and dilated, the left also but to a smaller degree. The tricuspid, pulmonic, and aortic valves were delicate and competent. The mitral ring was somewhat dilated, and the contact surfaces of the cusps were slightly thickened and rolled. The chordae tendineae were shortened. The interventricular and interauricular septums were intact.

Fig. 2.

When the great vessels were opened a defect was seen in the aortic septum 10 mm. above the margins of the pulmonic valves. The opening measured 12 mm. in diameter, and the edges were smooth (Fig. 4). Examination of the lungs showed small areas of atelectasis and moderate congestion. Microscopic examination showed the mitral valve cusps to contain a relatively large amount of cellular collagen tissue and to be moderately thickened. There were no signs of acute inflammation. There were no recent or old inflammatory changes about the margin of the aortic septal defect. The remainder of the microscopic examination was negative.

Comment: At the time of performance of the thoracic aortography on this patient we were using a manual cassette changer. Only one, or at most two, exposures had been found to be practical. Had we then had the automatic

camera which we now use, it might have been demonstrated that the Diodrast left the aorta and entered the pulmonary circuit at a low level of the ascending aorta, and the diagnosis would have been apparent. As it was, there was some uncertainty as to the exact location of the shunt.

Fig. 3.

The mechanism of death in this child can only be inferred. Too ardent "supportive" fluid and transfusion therapy, which is a not uncommon cause of death in cardiac operations in children, was probably not a factor, as she received a total of only 275 c.c. of pre- and intraoperative fluid and blood. The operative shock itself was probably the determining factor. That surgery of this nature should not be performed in such a "poor risk" patient is an objection which will always rise to plague the surgeon and the referring physician. It can be said in justification that a bleak outlook for life and comfort may be weighed against the risk of operation; if the underlying condition is believed to be remediable, one is justified in assuming the risk.

DISCUSSION

The ages of the fourteen patients with proved aortic septal defect have ranged from 3 days to 37 years. Two died in the first week of life, seven during the first year, three from one year to nine years, two in the third decade, and two in the fourth. The sex of the patients was noted in eleven instances; five were male and six female. The symptomatology and physical findings were variable in the group, but in general it might be said that no characteristic picture can be drawn. Cyanosis was present in eight cases and varied from mild and transient

Fig. 4.

to marked and continuous. The two patients with the most marked cyanosis had also clubbing of the fingers and toes. Dyspnea was noted in the protocols of nine patients. In two cases it was terminal, but in the others it was of significant duration. Disturbed cardiac action was mentioned in three cases in the literature and was described as "irregular" in one, "violent" in another, and "gallop" in the third. The subject of the present report had auricular fibrillation of unknown duration. Disturbances of compensation were present in seven patients. Edema appeared late in five, was confined to the lower extremities in

four, and was generalized in one. Murmurs were noted to be absent in three cases, two infants of 3 days and 5 days and a 9-year-old child. There was no mention made of a murmur in three additional cases. Each of the remaining eight patients presented a systolic murmur, and four had also a diastolic murmur. In one case[8] a systolic pulmonic murmur was present at the age of 20 years but could no longer be heard at the age of 22 years. Ordinarily the systolic murmur was described as being most intense in the pulmonic region. The double murmurs were merely described as "loud" in one case. In two cases they were most intense in the second and third left interspaces. In the patient described above there were murmurs suggestive of a patent ductus arteriosus and, at the apex, those suggestive of mitral stenosis. The microscopic examination of the valve cusps did show some changes consistent with an old rheumatic infection.

Death was due to disturbances of compensation in six cases, to tuberculosis in one, to *Bacillus mucosa capsulatus* septicemia in one, to pulmonary hemorrhage in one, and to operation in one.

At autopsy the right ventricle was hypertrophied in ten cases in which a description of the heart was adequate. In four cases the left ventricle was also hypertrophied. Associated cardiac defects were found in six instances, including perforation of the pulmonary valves, patency of the ductus arteriosus, patency of the foramen ovale, bicuspid aortic valve with an interventricular septal defect, and origin of the right pulmonary artery from the aorta. The ascending aorta was noted to be enlarged in two cases, contracted at the arch in one. The pulmonary artery was larger than normal at the base in one case. The aortico-pulmonic communication varied in size from 2 to 16 mm. in diameter and was situated usually in the first few centimeters above the valves, but occurred at the level of the pulmonic valve in one case and midway between the valve and the innominate artery in another. In shape it was round to oval, and the edge was smooth. In no case was mention made of any intimal change which might have been due to inflammation. In Fisher's[10] case there were large vegetations attached to segments of the aortic and pulmonic valves, but the defect was unaffected.

Clinically the defect might be suspected in the presence of an enlarged heart with right ventricular preponderance, disturbances of compensation, and either a systolic murmur or systolic and diastolic murmurs at the pulmonic area. Roentgenologic examination has been reported in only one case in the literature. Erf's[13] patient had a normal cardiac configuration. Our patient, on the other hand, showed marked dilatation of the pulmonary vessels, hilar dance, dilatation of the pulmonary artery, and generalized cardiac enlargement. The electrocardiogram in Erf's case showed right axis deviation with notching and spreading of the P waves. Our patient had a marked right axis deviation. The RST segment was depressed in Leads II and III, the T waves were flat in Lead I. Cardiac catheterization would be of diagnostic value if, as in Dexter's[20] case, the catheter passed through the defect into the aorta from the pulmonary artery. Otherwise, it would merely give evidence of an arteriovenous shunt and would not differentiate the condition from a patent ductus arteriosus. Angiocardiography might be expected to be of diagnostic aid in rare cases. It was unsuccessful in that of

Erf. Usually the dye would not pass from the pulmonary artery into the aorta because of pressure difference, and it would be difficult to demonstrate the dye passing from the aorta into the pulmonary circuit after circuit of the lungs and left heart. Thoracic aortography remains as the diagnostic procedure of choice when the condition is suspected. The study in our experience has been without ill effects and has been done at all ages. There have been no untoward reactions from the mechanics or from the 70 per cent Diodrast.

The surgical correction of this condition, although rather formidable, seems entirely possible, and no doubt will be accomplished in time.

SUMMARY

An unusual malformation of the great vessels present in a 9-year-old child has been described, together with a review of the available findings in the thirteen cases hitherto reported. Thoracic aortography is suggested as the diagnostic method of choice when the lesion is suspected.

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AURICULAR CALCIFICATION

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THE occurrence of auricular calcification of sufficient magnitude to be radiographically visualized is, in general, considered infrequent. In a review of the recent literature, auricular calcification visualized on a roentgenogram has been reported in only six patients. Nevertheless, in a period of ten years at the Goldwater Memorial Hospital, such calcification has been observed in eight cases. Post-mortem verification was obtained in five patients. Two are living. Permission for post-mortem study was refused in one.

Scholtz[1] reviewed the literature in 1924 and reported thirty cases of cardiac calcification. Of these, three patients had calcification of the left auricle. Two of the three had additional sites of abnormal calcium deposits (lungs, kidneys, and elsewhere). The third case was described by Oppenheimer[2] in 1912, and was a patient with a mitral stenosis, in whom widespread calcification of the left auricular endocardium was found post mortem. Observations of auricular calcification were reported by other observers,[3,9,18] a total of twelve cases. In none of these case reports were roentgenograms presented. VonGlahn[6] found one case of left auricular calcification in thirty-one rheumatic hearts seen post mortem, while Gross[7] and Koletsky[18] reported one case in eighty-seven and one case in 100 rheumatic hearts autopsied by each, respectively.

The first radiological demonstration of auricular calcification was described by Shanks, Kerley, and Twining (1938)[10] in a patient with mitral valvular disease. Begg,[11] in 1944, demonstrated a calcified left auricle, and calcification of the aortic and mitral valves. Schwedel[12] has an illustration of one in his book, while Epstein[13] has reported three patients. In the above six patients with positive radiological findings no post-mortem findings were mentioned.

This presentation includes eight cases, five with roentgen and necropsy findings of left auricular calcification and three with roentgen findings only.

CASE REPORTS

CASE 1.—F. P., a 51-year-old white man, was well until the age of 31 years, when he began to complain of dyspnea on effort. At that time his physician told him that he had a "rheumatic heart." He was digitalized then and continued to take digitalis for the rest of his life. Exertional dyspnea became progressively worse and signs of chronic cardiac failure slowly developed. From October, 1945, until his death in November, 1948, the patient spent most of his time hospitalized with chronic left-sided congestive failure. During the last eighteen months of his life, frequent

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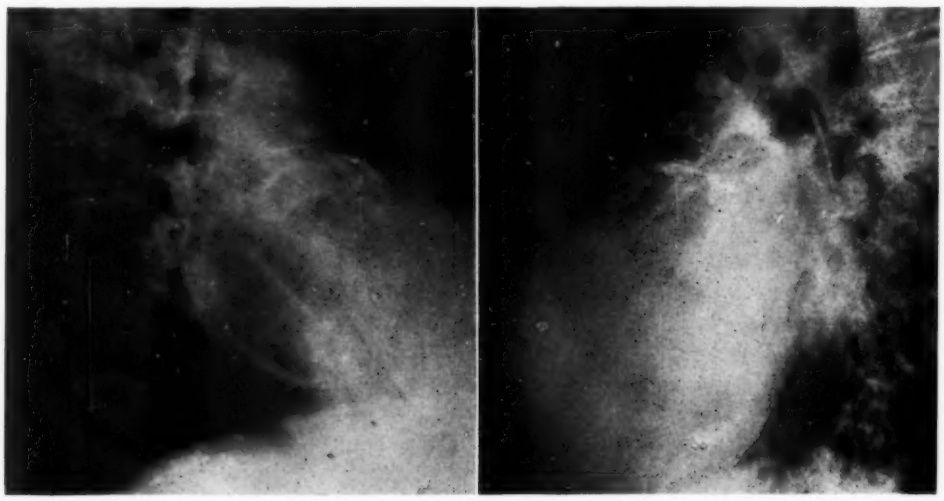
thoracenteses and abdominal paracenteses were required. Physical examination of the heart revealed the point of maximum impulse to be in the sixth intercostal space in the anterior axillary line. Systolic and diastolic murmurs and thrills were present at the base. Auricular fibrillation began three years before death.

Roentgenographic examination revealed a markedly enlarged heart with a transverse diameter measuring 26 cm. The anteroposterior diameter of the heart measured 17 cm. A large circular calcific deposit was present within the central portion of the cardiac silhouette in the anteroposterior projection, and in the posterior portion in the lateral projection. The diameter of this calcific deposit measured 10 cm.

At post-mortem examination the heart weighed 700 Gm. and showed right and left ventricular hypertrophy. Both auricular cavities and the right ventricular cavity were dilated. There was rheumatic valvulitis (active and inactive) with stenosis and insufficiency of the mitral and tricuspid valves. Aortic valvulitis had produced insufficiency only. The left auricle contained a large friable thrombus which filled the greater portion of the cavity and was attached over a 6 by 4 cm. area. Almost the entire endocardium of the left auricle was calcified. No other areas of abnormal calcification were found elsewhere in the body.



A B.

Fig. 1.—Roentgenograms of J. K. (*A*) and S. H. (*B*) showing areas of calcification in the left auricle verified at necropsy.

CASE 2.—J. K., a 73-year-old white man, was well until August, 1944, when he developed increasing shortness of breath and ankle edema. Auricular fibrillation was noted at that time. There was no antecedent history of rheumatic fever. His blood pressure was 140/80 mm. Hg. His heart was enlarged to the left and a loud systolic murmur was heard over the entire precordium. In November, 1946, he developed signs of intestinal obstruction and expired twenty-four hours later.

Roentgenographic examination revealed a markedly enlarged heart with a transverse diameter measuring 23 cm. The cardiac configuration was that of a combined lesion. The posteroanterior projection revealed a large circular calcific deposit in the central portion of the cardiac silhouette which measured approximately 10 cm. in diameter. In the lateral projection this calcific deposit was situated posteriorly (Fig. 1, *A*).

At post-mortem examination the heart weighed 850 Gm. The ventricles were hypertrophied and the chambers dilated. There was an old rheumatic valvulitis of the mitral valve with stenosis

and insufficiency, and an old aortic valvulitis with calcification. There were two small calcified nodules on the auricular surface of the mitral valve. The entire lumen of the left auricle was lined by a large, calcified shell. On the surface of this shell was a mass of thrombotic material largely filling the left auricular cavity. Moderate coronary atherosclerosis was present. Microscopic study of the left auricle showed the endocardium to be markedly thickened and hyalinized with calcification of the center of the hyalinized tissue. There were only islets of auricular muscle tissue, the rest of the wall being made up of connective tissue. There were no other abnormal areas of calcification found at post-mortem examination. In addition, there was also found a generalized fibrinopurulent peritonitis secondary to a perforation of the sigmoid, which was involved by an adenocarcinoma.

CASE 3.—S. L., a 56-year-old white woman was told she had a "bad heart" during pregnancy in 1916 at the age of 26 years. In 1927, she suffered a right hemiplegia, which was thought to have been caused by a cerebral embolus. In 1930, she first developed exertional dyspnea and orthopnea. From 1940 until her admission to this hospital in July, 1944, she had several hospitalizations for congestive failure. No history of rheumatic fever was obtained. When admitted, she was in right- and left-sided heart failure and auricular fibrillation was present. Her blood pressure was 130/80 mm. Hg. Systolic and diastolic murmurs and thrills were present at the apex and a systolic murmur was heard at the base. In spite of therapy she remained in chronic congestive failure until her death in September, 1946.

Roentgenographic examination revealed a generalized enlargement of the heart with a short, curvilinear, calcific deposit in the upper portion of the left half of the cardiac silhouette, below the middle cardiac segment. There was a small effusion at the right base.

At post-mortem examination the heart weighed 570 Gm. The left and right ventricles were only slightly hypertrophied. Both auricles were dilated. In the left auricular appendage was a large adherent thrombus. The endocardial wall of the auricle beneath the thrombus was calcified. Diffuse fibrosis was present throughout the auricular myocardium. The mitral valve was markedly thickened and the mitral orifice was stenosed and insufficient. The mitral ring contained calcium deposits. A moderate tricuspid valvulitis was also present. The coronary vessels were all widely patent. No other abnormal areas of calcification were found.

CASE 4.—F. B., a 49-year-old white man, was admitted to this hospital in August, 1946, and died in June, 1948. He was well until September, 1934, when he developed dyspnea, orthopnea, and ankle edema. There was no history of rheumatic fever or hypertension. Auricular fibrillation was continually present from 1934 to his death. He had had a dozen hospital admissions for congestive failure in ten years. In 1945, he had an episode of precordial pain with electrocardiographic evidence of posterior wall infarction. The heart was enlarged to the left. There were apical and basal systolic and diastolic murmurs. His cardiac failure responded well initially. He had several recurrences of congestive failure and died in June, 1948.

Roentgenographic examination revealed a generalized enlargement of the heart. It was triangular in shape and had the configuration of a "mitral" heart. A curvilinear, calcific deposit with its convexity to the right was present within the upper portion of the right half of the cardiac silhouette. This calcification was situated 2.5 cm. to the right of the spine and about 2.5 cm. above the diaphragm.

At post-mortem examination the heart weighed 640 Gm. All of the chambers were moderately dilated. The mitral cusps were fused, thickened, and calcified. The mitral and aortic orifices were stenosed. The aortic valves presented evidence of valvulitis and there was a deposit of calcium in the cusps. Similarly, tricuspid stenosis was present, but without calcium in the cusps. A large thrombus adherent over a 7 by 5 cm. area was present in the left auricle. There was a deposit of calcium in the left auricular endocardium. The right ventricle was hypertrophied. The coronary vessels showed moderate to advanced atherosclerosis in all major branches. Except for left renal lithiasis in a pyelonephritic kidney, there was no other evidence of abnormal calcification.

CASE 5.—S. H., a 52-year-old white woman, was admitted to this hospital in November, 1939. She had had scarlet fever at the age of 9 years and several bouts of hemoptysis at the age of 14 years, which she said were caused by "heart disease." She was on limited activity after that.

At the age of 30 years, she had a cerebrovascular accident with a residual right facial weakness and a partial aphasia. Since that time she complained of occasional precordial pain, palpitations, and dyspnea on exertion. From 1932 on, she had recurrent bouts of cardiac failure. Physical examination revealed a blood pressure of 130/90 mm. Hg. The point of maximum impulse was in the anterior axillary line. A diastolic thrill was felt at the lower left sternal border, and systolic and diastolic murmurs were heard in this area. During her stay at this hospital, she was in cardiac failure frequently. Auricular fibrillation and auricular flutter were present on different occasions. The patient died in February, 1949.

Roentgenographic examination revealed a generalized enlargement of the heart. The cardiac silhouette was triangular in shape with an accentuation of the left middle cardiac segment. The configuration was that of a "mitral" heart. There were several irregular calcific deposits within the upper left half of the cardiac silhouette above the junction of the base of the left ventricle with the middle cardiac segment. The lateral projection showed this to be situated in the posterior half of the cardiac silhouette (Fig. 1, *B*).

At post-mortem examination the heart weighed 420 Gm. The atria were large and distended. The endocardium of the left atrium showed plaques of calcification and a few calcified verrucae. The mitral orifice was narrowed with marked wrinkling, scarring, and thickening of the mitral cusps. The tricuspid orifice showed some stenosis with thickening and wrinkling of the valve edges. The myocardium was somewhat hypertrophied. The coronaries showed slight atherosclerotic changes. Microscopic examination showed the mitral and tricuspid valves to be markedly thickened and fibrotic, but showed no areas of calcification, cellular infiltration, or vascularization.

Case 6.—W. S., a 66-year-old man born in Jamaica, was admitted September, 1943, with complaints of dyspnea, orthopnea, and ankle swelling. His history revealed an episode of generalized edema and joint pain at the age of 40 years. At the age of 52 years, he developed anorexia and exertional dyspnea and fatigued easily. Prior to his admission here, he had been hospitalized seven times for congestive failure. His blood pressure was 130/70 mm. Hg. There was evidence of right- and left-sided heart failure. The point of maximum impulse was in the fourth intercostal space in the midclavicular line. A systolic thrill was present at the apex. A harsh systolic murmur was heard over the whole precordium, loudest at the mitral area. A soft early diastolic murmur was present at the apex. A harsh systolic murmur, transmitted to the neck, was loudest at the aortic area. The clinical diagnosis on admission was rheumatic heart disease, mitral insufficiency and stenosis, aortic stenosis, and auricular fibrillation. His electrocardiogram showed auricular fibrillation, premature ventricular contractions with coupling, and digitalis effect. In spite of active cardiac therapy, the patient had recurrent episodes of congestive failure from the time of admission until his death in May, 1949. Autopsy permission was refused.

Roentgenographic examination revealed a generalized enlargement of the heart. The cardiac silhouette was triangular in shape. The configuration was that of a combined lesion. The posteroanterior projection revealed a curvilinear calcific deposit in the central portion of the cardiac silhouette. In the lateral projection, this calcific deposit was situated posteriorly. This calcification was within the area of the left auricle.

Case 7.—L. L., a 51-year-old white woman, was admitted in May, 1946, for a gastroenteritis and nasopharyngitis. Her history revealed chorea at the age of 12 years. At the age of 17 years she became ill, the main complaints being pain in the cervical spine, weakness, loss of energy, ankle edema, and dyspnea on exertion beginning in 1931. She was treated in a cardiac clinic after 1932, with a diagnosis of rheumatic heart disease. On physical examination the heart was enlarged to the right and left. The rhythm was irregular. A soft apical diastolic murmur was heard. A soft diastolic murmur was also heard at Erb's point. The clinical diagnosis has been rheumatic heart disease, enlarged heart, mitral insufficiency and stenosis, with auricular fibrillation. Electrocardiograms showed auricular fibrillation and right axis deviation. In June, 1946, her serum calcium was 9.8 mg per cent and serum phosphorus was 3.0 mg per cent. She was discharged to the cardiac clinic in September, 1946. However, she subsequently moved away and changed her home address, so that further follow-up is not available.

Roentgenographic examination revealed enlargement of the heart. The cardiac silhouette was triangular in shape. The configuration was that of a "mitral" lesion. The posteroanterior

projection showed an irregular calcareous deposit in the upper left half of the cardiac silhouette below the middle cardiac segment, probably within the left auricular appendage. In the lateral and oblique projections there was observed an additional semicircular curvilinear deposit in the posterior half of the cardiac silhouette, in the region of the left auricle (Fig. 2,*A*).

CASE 8.—L. R. was a 59-year-old white woman who had had three admissions to this hospital in 1941 and 1942 for congestive failure. Her history revealed rheumatic fever at the age of 5 years. The following year she had scarlet fever and painful, tender joints which cleared in one month. Following this, she had several attacks of polyarthritis, and at 8 years of age she had chorea. Since then she has had frequent attacks of dyspnea, palpitations, and orthopnea. Physical examination showed the heart to be enlarged to the right and left. Systolic and diastolic murmurs were present at the apex. The rhythm was totally irregular and the blood pressure was 136/80 mm. Hg. Her electrocardiogram showed auricular fibrillation, no deviation of the electrical axis, and occasional ventricular premature contractions. The clinical impression was that she had rheumatic heart disease with an enlarged heart, mitral and aortic insufficiency and stenosis, and auricular fibrillation. Further observations after her discharge in April, 1942, are not available.

A. B.

Fig. 2.—Roentgenogram of L. L. (*A*) in right oblique position shows a curvilinear calcification in the left auricle. Patient S. R. (*B*) shows an area of calcification in the left half of the cardiac shadow in the P-A projection which probably represents auricular calcification.

Roentgenographic examination revealed a generalized enlargement of the heart to the right and to the left with an accentuation of the left middle cardiac segment. The cardiac configuration was that of a "mitral" heart. The posteroanterior projection showed a curvilinear calcific deposit present in the left half of the cardiac silhouette (Fig. 2,*B*). The lateral and oblique projections localized this calcification posteriorly within the region of the left auricle. There was a small pleural effusion at the right base.

DISCUSSION

Abnormal deposits of calcium may occur in patients receiving massive doses of Vitamin D, or in patients with abnormal calcium metabolism. In our cases,

TABLE I. FIVE CASES WITH POST-MORTEM EXAMINATIONS

NAME	AGE	SEX	RHYTHM	VALVES INVOLVED	ADDITIONAL CARDIAC CALCIFICATIONS	EXTRACARDIAC CALCIFICATIONS	LEFT AURICULAR MURAL THROMBUS
F. P.	51	M	Auricular fibrillation	Mitral stenosis and insufficiency; aortic insufficiency; tricuspid stenosis and insufficiency	None	None	Yes
J. K.	73	M	Auricular fibrillation	Mitral stenosis and insufficiency; aortic valvulitis	Aortic and mitral cusps	None	Yes
S. L.	56	F	Auricular fibrillation	Mitral stenosis and insufficiency; tricuspid valvulitis	Mitral annulus	None	Yes
F. B.	49	M	Auricular fibrillation	Mitral stenosis and insufficiency; aortic stenosis and insufficiency; tricuspid stenosis	Aortic and mitral cusps	Staghorn calculus, left kidney	Yes
S. H.	52	F	Auricular fibrillation	Mitral stenosis and insufficiency; tricuspid stenosis	None	Medullary region, left kidney	No
				Three Cases Without Post-Morten Examination			
W. S.	66	M	Auricular fibrillation	Mitral stenosis and insufficiency; aortic stenosis	None		
L. L.	51	F	Auricular fibrillation	Mitral stenosis and insufficiency	None		
L. R.	59	F	Auricular fibrillation	Mitral stenosis and insufficiency; aortic stenosis and insufficiency	None		

there was associated rheumatic heart disease with no history of Vitamin D therapy or apparent disturbance in calcium metabolism.

VonGlahn[6] reviewed thirty-one cases of rheumatic valvulitis found post mortem. Nine cases had auricular endocarditis, one of which had calcium deposition in the auricular endocardium. He considered the calcification to be secondary to the previous inflammatory changes and stated that "it is probable that many of the plaques of calcium found in the auricular endocardium in chronic mitral disease mark the sites of previous acute rheumatic lesions." Gross[7] made post-mortem studies on eighty-seven patients with rheumatic heart disease and observed macroscopic auricular lesions in 80 per cent and microscopic evidence of rheumatic involvement of the left auricle in every case. In only one case was there a deposition of calcium in the left auricular endocardium.

All of our cases had long-standing rheumatic heart disease, and it seems likely that the auricular calcifications were the end result of rheumatic auriculitis.

In our group, there were four men and four women (Table I). Their ages ranged between 49 and 73 years, with an average age of 56 years. Auricular fibrillation was present in every one. In the five cases with post-mortem verification, the mitral valves were involved in all, showing stenotic and insufficiency lesions. The aortic valve was involved in three cases (Cases 1, 2, and 4). The tricuspid valve was involved in four cases (Cases 1, 3, 4, and 5). The three cases without post-mortem studies had mitral stenosis and insufficiency. Aortic valvular disease was present in two.

Of the five cases with necropsy findings, three demonstrated additional intracardiac calcifications. Case 3 showed calcification of the mitral annulus and Cases 2 and 4 showed calcification of the aortic and mitral cusps. A large staghorn calculus was found in the left kidney of Case 4, while "calcification in the medullary region of the right kidney" was reported in Case 5.

Mural thrombosis in the left auricle was found in four of the five patients which came to post-mortem examination. One did not demonstrate this finding.

There may be causes for auricular calcification other than rheumatic disease. Calcium may be deposited in dead or devitalized tissue. Numerous reports have appeared demonstrating ventricular calcification in areas of myocardial infarction.[16,17] Cardiac calcification may occur after an antecedent myocarditis. Berk[15] has reported a case with rheumatic heart disease and calcification of a mural thrombus in the left auricle. Diamond[14] reported a 26-week-old premature infant with calcification of both ventricles and the right auricle which he thought was secondary to degeneration of muscle fibers. Our findings would indicate that the presence of left auricular calcification, in the absence of any endocrine or vitamin disturbance, presupposes that the patient has rheumatic heart disease with a calcified auricular endocardium (or thrombus) secondary to auriculitis.

Left auricular calcification is usually curvilinear in outline and within the area of the left auricle. Valvular deposits (occasionally associated with auricular deposits) at times may be recognized fluoroscopically. Coronary artery calcifications generally are superficial, linear, and small, and follow the distribution of the coronary vessels. Ventricular calcifications generally occur in the apical region but may occur elsewhere. Pericardial calcifications occur most commonly

along the diaphragmatic surface of the heart and anteriorly over the right ventricle, but they may be generalized. Extracardiac calcific deposits in the costal cartilages and mediastinal structure are readily differentiated from intracardiac calcifications.

SUMMARY

Eight cases of left auricular calcification are reported, five with post-mortem studies. All had rheumatic heart disease. Although not a common finding, calcification of the left auricle or its appendage is not rare and is generally secondary to a rheumatic auriculitis.

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Clinical Reports

CONGENITAL MITRAL ATRESIA, TRANSPOSITION OF THE GREAT VESSELS, AND CONGENITAL AORTIC COARCTATION

A Case Report and an Interpretation of the Anomaly

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CASES of congenital mitral atresia are rarely seen. Abbott[1] lists only 5 out of 1,000 cases of congenital heart anomalies. In an analysis of 27,077 deaths at the Charity Hospital of New Orleans,[2] not a single instance of this anomaly was found. Numerous other reports attest to the rarity of this defect.[3-11] In a review of the literature,[12-33] forty-eight cases of atresia or absence of the mitral orifice were found. An analysis of the associated characteristics and anomalies of forty-one of these cases together with those of the present case are given in Table I. From a study of this table it is seen that in a large majority of the cases the left side of the heart is poorly developed while the right chambers are hypertrophied. The foramen ovale, the ductus arteriosus, and the interventricular foramen are usually open. In 42.5 per cent of the cases there was an associated transposition of the great vessels. In Table II the age and sex variations in this series of cases are shown. The anomaly is more frequent in males than in females in a ratio of 3 to 2. Over one-half of the patients died during the first week of life; two-thirds lived less than one month; while almost all died before the age of 10 years.

The present case not only exhibits the rather rare congenital mitral atresia in a triloculate heart, but it shows other anomalies as well. While the abnormalities reported here are not new in themselves, the peculiar combination in which they occur in a single subject appears sufficiently unique to warrant a description. In addition, the present case has suggested a theory of interpretation which may explain many of these cases.

CASE REPORT

The patient was a 6½-pound Negro male infant born spontaneously on March 23, 1949, following an uneventful pregnancy and labor. The mother was a woman of 36 years with a history of five previous normal pregnancies. These five children are alive and well, their ages ranging from 2 to 10 years. There are no evident congenital malformations among any of the children with the exception of partial albinism in a boy aged 6 years. There is no history of

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TABLE I. TABULATION OF THE OTHER ANOMALIES ASSOCIATED WITH FORTY-TWO CASES OF CONGENITAL MITRAL ATRESIA*

CHARACTERS	PRESENT	ABSENT	NOT MENTIONED	PER CENT PRESENT
Closed or absent mitral valve	42	0	0	100.0
Defective left ventricular development	31	1	10	96.9
Small left atrium	32	2	8	94.1
Open foramen ovale	32	5	5	86.5
Large right atrium	31	3	8	91.2
Large right ventricle	30	1	11	96.8
Interventricular septal defect or missing septum	33	4	5	89.2
Aortic atresia or hypoplasia	21	18	3	53.8
Transposition of the great vessels	17	23	2	42.5
Open ductus arteriosus	24	12	6	66.6

TABLE II. SEX AND AGE DISTRIBUTION: ANALYSIS OF FORTY-TWO CASES*

	NUMBER PRESENT	SEX OR AGE NOT GIVEN	PER CENT PRESENT
Males	20	9	60.6
Females	13		39.4
Age less than 1 week	19		51.3
Age less than 1 month	24	5	64.4
Age less than 10 years	35		94.6
Age more than 10 years	2		5.4

*In both tables the per cent present is calculated on the basis of the total number of cases in which a definite statement regarding the presence or absence of the various characters could be found in the literature. Of the forty-eight cases with congenital mitral atresia found, seven[18,25] were not sufficiently well characterized to permit their inclusion in the above study. The tables include the case described in this paper.

syphilis in either parent. No history of congenital heart disease in the families of either parent was elicited. No history of exanthematous or other disease of the mother in the first two to three months of pregnancy could be obtained. The delivery occurred in the home. The parents and the attending physician stated that the child cried normally and no cyanosis was noticed. There is no record of auscultation of the heart. After about thirty-three hours, the mother noticed that the child was gasping for breath and that his nail beds had become blue. Approximately three hours later the child was pronounced dead by the local physician. At this time he observed that the liver was markedly enlarged, its free edge reaching well into the right lower abdominal quadrant. Two days later the infant was received in this department where the abnormality of the heart was discovered. Aside from the conditions to be described in the heart, the pathologic findings were limited to hepatic and pulmonary congestion and the usual findings in anoxemic death. There was a partial atresia of the right ureter 1 cm. below the pelvis of the kidney.

The appearance of the heart: The heart was unusually large (Fig. 1). Its greatest transverse diameter was 6.8 cm. while that of the thorax at the same level was 8.5 cm. The heart was not weighed since a close study of the great vessels precluded its removal. The right atrium was markedly dilated and filled with clotted blood. It made up the entire heart to the right of the midline and measured 2.3 cm. transversely and 4.3 cm. longitudinally. At its cranial border the atrium presented a relatively large auricle measuring 1.5 cm. in both transverse and longitudinal directions. A common ventricle made up the entire ventral aspect of the heart to the left of the

midline. The ventricle was pear-shaped and its apex pointed laterally. The distance from the apex to the coronary sulcus was 4.5 cm. The left atrium was considerably smaller than the right, measuring 1 cm. transversely and 2 cm. longitudinally. It could be seen only after the apex of the heart had been displaced cranially, for it was completely covered by the ventricle. At the left lateral border of the atrium was a minute auricle, 0.6 by 1 cm. in size. The right atrium received the superior and inferior venae cavae in the normal manner. The right and left pulmonary veins entered the small left atrium as single trunks 0.5 cm. apart.

The interior of the heart: There was nothing unusual in the mode of entrance of the caval vessels into the right atrium. On the inner wall of the right atrium was a well-marked crista terminalis. The Eustachian and Thebesian valves were normal. The coronary sinus opened normally into the right atrium. There was a prominent limbus fossae ovalis and torus Loweri. The fossa ovalis measured 1.3 cm. in diameter, while the foramen ovale was a slit about 0.5 cm.

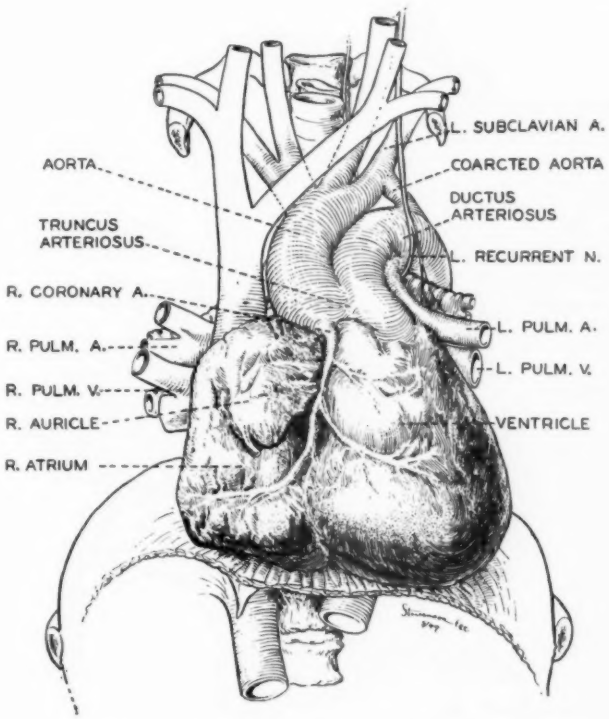

Fig. 1.—The appearance of the heart. The first and eighth ribs are shown on either side to demonstrate the enlargement of the heart through comparison with the width of the chest wall. The part labeled truncus arteriosus is the proximal 0.4 cm. of the exit tube which is not separated externally.

long and 0.3 cm. high at the craniodorsal aspect of the interatrial septum. The tricuspid orifice was very large, measuring 6.6 cm. in circumference. There was a fibrous anterior cusp; the medial and inferior cusps were fleshy and nodular. The latter was small and lay at the caudal margin of the sagittally placed tricuspid orifice. The medial cusp was attached to the dorsal wall of the heart.

The ventricle showed evidence of considerable muscular hypertrophy. The cortical layer of its wall measured 0.3 cm. in diameter, while the trabecular layer varied from 0.4 to 0.5 cm. in thickness. Two of the muscular trabeculae were hypertrophied, forming prominent ledges on the cardiac interior. In Fig. 2 these are labeled Muscle Ledges A and B. Muscle Ledge A extended in a ventrolateral direction from the region of the dorsal wall of the heart near an aortic semilunar

valve (*A* in Fig. 2) and passed cranial to the anterior cusp of the tricuspid valve to reach the ventral wall of this chamber. Muscle Ledge B, beginning in close relationship with Muscle Ledge A, coursed along the dorsal wall of the ventricle in a caudal direction presenting a free crescentic margin directed toward the tricuspid orifice. It became indistinct on the lowermost portion of the inner ventricular wall. Arising from the caudal end of Muscle Ledge B was a threadlike structure which crossed the ventricular cavity to become attached to the inner ventricular wall near the anterior leaf of the tricuspid valve.

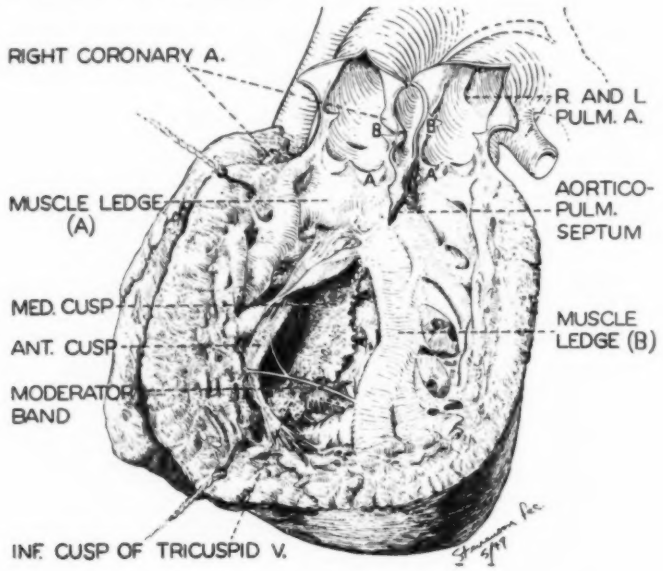

Fig. 2.—Interior of the ventricle. *Muscle Ledge A* has been so named because proximal bulbar Ridge A is postulated to have formed this structure. Normally it represents the contribution of proximal bulbar Ridge B to the supraventricular crest of the right ventricle. *Muscle Ledge B* is formed in this case by proximal bulbar Ridge B. It represents the septal muscle band, or the trabecula septomarginalis normally formed by Ridge A. The aorticopulmonary septum is seen to be complete. Observe the left coronary orifice on the dorsal aortic wall. In the text semilunar valves *A* and *B* and *A'* and *B'* have been called the septal valves.

The walls of the diminutive left atrium were smooth except in the region of the auricle. The two pulmonary veins entered the dorsal wall of the atrium as separate trunks 0.5 cm. apart. The left atrium was about one-tenth the size of the right atrium. The mitral orifice was completely absent. No scarring, puckering, or dimpling, usually associated with an atretic mitral orifice, was present. No valve leaflets were identifiable. Cuts made in this region in an effort to locate a slitlike left ventricle among the maze of trabecular formations in the dorsal wall of the heart below the left atrium failed to reveal any evidence of its presence. Microscopic sections gave no evidence of endocarditis or myocarditis.

The great vessels: The vessels arose from the ventricle at the cranial end of its left border (Fig. 1). For a distance of 0.4 cm. cranial to the ventricle the vessels were incompletely separated. The aorta was to the right and somewhat ventral to the pulmonary artery. The line of pericardial reflection from the vessels lay 1.5 cm. cranial to the ventricle. Under cover of a large thymus gland, the ascending aorta gave rise to a short innominate artery, a left common carotid artery, and a left subclavian artery. The innominate artery branched in the standard manner. The pulmonary trunk divided into right and left branches just distal to the line of pericardial reflection. The trunk then continued as an arching vessel which was, in turn, continuous with the descending aorta. This arch was the widely patent ductus arteriosus. A connecting channel much smaller in diameter than either of the great vessels joined the highest point of the ductus

with the origin of the left subclavian artery. The recurrent branch of the left vagus hooked around the caudal margin of the ductus and ascended at a more dorsal level in the usual manner. The aortic and pulmonary trunks each had three semilunar valves. In each of the trunks, two valves were adjacent to the aorticopulmonary septum. In Fig. 2, these have been labeled *A* and *B* and *A'* and *B'*, respectively. Above the aortic valves *A* and *B*, the coronary orifices may be seen. Because of an excessive rotation of the bulbus cordis during the formation of the heart (to be fully considered later) the right coronary artery arose ventrally above valve *B*. The left coronary artery then arose dorsally above valve *A*. The right artery ran in the coronary sulcus for a short distance before giving off its posterior descending branch (Fig. 3). Due to the rotation mentioned above, however, this branch was on the ventral surface of the heart. In the same manner, the left coronary artery sent its anterior descending branch along the dorsal surface of the heart toward the apex. The aorticopulmonary septum was shared by the two great vessels from its caudal free margin well within the ventricle to a point 0.4 cm. distal to the exit of the vessels from this chamber. The circumferences of the aorta and the pulmonary arteries at the semilunar valves measured 1.8 and 2.1 cm., respectively.

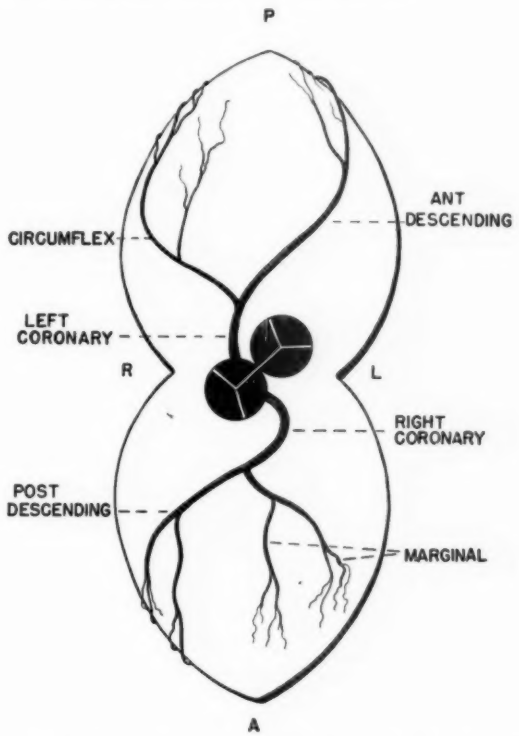

Fig. 3.—The course of the coronary arteries. The ventricle has been divided in a frontal plane from the apex toward the base. The base of the ventricle is pictured as devoid of relationship with the atria. The ventral surface is below while the dorsal surface is above. These are indicated by the letters *A* and *P*. The letters *R* and *L* indicate the right and left sides.

The cause of death: The direction taken by the circulation during intrauterine life was such that there was no interference with the oxygen supply of the embryo. Blood entered the enlarged right atrium through the systemic veins, the coronary sinus, and the foramen ovale. It passed through the tricuspid orifice into the ventricle, where it was distributed to the head and upper extremities by the transposed aorta, and to the lungs and to the remainder of the body by the enlarged pulmonary trunk and ductus arteriosus, Following birth the unoxygenated blood,

reaching the right atrium via the systemic veins and the coronary sinus, mixed freely with the oxygenated blood returning from the lungs by way of the left atrium and the foramen ovale. The fact that the right and the left pulmonary arteries branched from the pulmonary trunk almost at right angles to its course provided a poor mechanical arrangement for the circulation of blood to the lungs. In addition, the minute size of the foramen ovale must have prevented any rapid return of oxygenated blood. There was no evidence of failure of full oxygenation of the blood going through the lungs; atelectasis was not present. It is thus easy to understand how cyanosis developed. The cyanosis, which was seen as a terminal event, demonstrates the presence of anoxemia, while the hepatomegaly attests to the existence of circulatory failure. The anatomic diagnosis was: mitral atresia of congenital origin, cor triloculare biatriatum, functional cor biloculare, absent interventricular septum, patent ductus arteriosus, congenital coarctation of the aorta at the aortic isthmus, and pure crossed transposition of the great vessels in situs solitus.

DISCUSSION

Theories of Etiology of Congenital Heart Disease.—Numerous causes have been listed for congenital cardiac anomalies. Among them are endocarditis, defective germ cells, adverse effects of the maternal organism, heredity and consanguinity, alcoholism, and syphilis.[34] Situs inversus is frequently associated with heart disease and has been used to explain various types of transposition.[35-37] In turn, many causes for situs inversus have been proposed.[38] New light has been thrown on the etiology of certain congenital anomalies by Gregg's[39] discovery of the role of maternal rubella in these cases. Anomalies involving mainly the eyes and the heart occurred in almost 100 per cent of the cases in which a history of maternal rubella in the first two to three months of pregnancy could be obtained. Dogramaci and Green[40] have summarized the various theories by saying that the proximate cause is arrested or imperfect development during intrauterine life. Both environmental and hereditary factors are listed among possible primary causes. In the present case no primary cause could be found. The proximate cause was imperfect development during the second month of embryonic life.

Etiology of Congenital Mitral Atresia.—Wieland,[41] in a discussion of tricuspid atresia, recognized the importance of the failure of shifting of the atrial canal to the right. Moenkeberg[22] attributed congenital mitral atresia to the displacement or maldirected growth of either the interatrial septum or the interventricular septum or both. In addition, he believed that shifting of the atrial canal played a role in the pathogenesis of some cases. Other theories have been based on the absence of the interventricular septum,[21] fetal endocarditis,[42] and overgrowth of endocardial cushion tissue.[6] Harris and Farber,[17] and others,[6] have attempted to explain the frequently associated anomaly of transposition of the great vessels by using Spitzer's theory.[43-45] A primary closure of the mitral valve, on the basis of either endocarditis or overgrowth of endocardial cushion tissue, is believed by these authors to set into motion the detorsion postulated in Spitzer's theory with reopening of the hypothetical right ventricular aorta. In the writer's opinion, cases with absent or rudimentary interventricular septa cannot be so explained, since, from a purely mechanical standpoint, there is no greater stimulus for the use and persistence of a right ventricular aorta than for the normal left one.

Embryologic Foundation for Interpretation of the Present Case.—Davis[46] traced the formation of the human heart from its paired anlagen to the period immediately before the onset of septation (fourth week). His diagrams show the development of the multiaxial primary cardiac bends establishing the right atrioventricular and the left bulboventricular sulci. The atrial canal lies at first to the left of the midline. Walmsley[47] called attention to the embryologic and phylogenetic association of the onset of septation in the atrium and in the exit tube. The shift of the atrial canal to the midline is related to its approximation with the bulbus cordis through the absorption of the bulboventricular spur.[48]

Much confusion is caused by the varied and overlapping embryologic and anatomic terms used in the region of the exit tube. In this discussion the bulbus cordis is considered to be divided into distal and proximal portions at the line of attachment of the semilunar valves. According to Walmsley,[47] the proximal bulbar portion is absorbed into the ventricle during development with the exception of that part which forms the conus arteriosus of the right ventricle. The distal bulbar portion becomes the intrapericardial portion of the truncus arteriosus. The ostium ventriculobulbare (Fig. 4) (hereafter called ostium V.B.) of Pernkopf and Wirtinger,[36] therefore, is between the ventricle and the proximal bulbar segment. The ostium bulbotruncare (hereafter called ostium B.T.) is between the distal bulbar segment and the truncus arteriosus. The truncus is divided by the septum between the fourth and sixth aortic arches into the aortic and pulmonary portions. This septum is continuous through the distal bulbar ridges with the proximal bulbar ridges. The latter are located in their respective portions of the bulbus. The distal bulbar ridges have been called Ridges I, II, III, and IV (Born), while the proximal bulbar ridges are called A and B (Boas) (Fig. 4). Ridge I is continuous with Ridge A and this ridge, as a whole, may be called Ridge I A. Ridges III and B are in similar arrangement, and together may be called Ridge III B. Ridges A and B are also called the sinistroventral and dextrodorsal conus ridges, respectively.[49] Ridges I and III are also called dextrodorsal and sinistroventral truncus ridges, respectively.[50]

In normal development the septal anlagen in the region of the truncus and the bulbus cordis develop with an initial spiral of 270 degrees[36,51,52] (Fig. 4.). In the process of bulbar absorption this spiral is reduced to 75 degrees. This is brought about through rotation of the bulbus. Counterclockwise torsion of 150 degrees occurs at the ostium B.T. (looking cranially from the ventricle) while 45 degrees clockwise torsion occurs at the ostium V.B. The minimal torsion at the latter point is explained by the fixation of the proximal portion of the bulbus to the atrial canal and to the ventricle. This is brought about through the fusion of Ridge A with the interventricular septum, and through the fusion of Ridge B with the medial and lateral cusps of the tricuspid valve, and the lateral wall of the right ventricle between the valve and the pulmonary artery. The latter is instrumental in forming the supraventricular crest of the normal right ventricle.

Interpretation of the Present Case.—In the present case the fixation of the bulbus to the atrial canal failed to occur. This permitted the canal to shift far to the right beyond its normal midline position. The septum primum approached the region of the atrial canal in the normal manner, but, due to the excessive

shifting of the atrial canal to the right, it came to lie over the region of the mitral orifice. Fusion then occurred between the septum primum, the fused dorsal and ventral endocardial cushions, and the lateral endocardial cushion. The mitral orifice was thus obliterated. This probably occurred during the sixth week of development.

As a second result of the failure of normal fixation of the proximal end of the bulbus, excessive clockwise torsion occurred at the ostium V.B. (looking cranially from the ventricle). The dorsal wall of the bulbus moved, therefore, to the right. This direction of movement coincides with that postulated to have occurred at the atrial canal. The two phenomena may, therefore, have been associated. Normally the clockwise rotation of 45 degrees of this region of the bulbus brings the aorta to the left and dorsal to the pulmonary trunk. In the present case, however, an additional 180-degree torsion occurred, placing the aorta in its right ventral position. The coronary arteries were also rotated to the same extent.

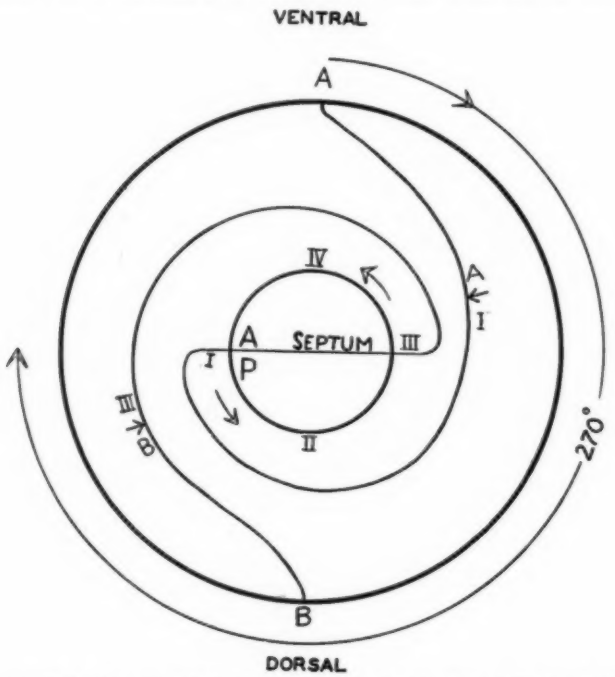

Fig. 4.—Schema of the interior of the bulbus cordis, looking cranially from the ventricle. The larger circle is the junction of bulbus and ventricle; the smaller circle is the junction of bulbus and truncus. Proximal bulbar ridges *A* and *B* spiral along the inner surface of the wall and are continuous with the distal bulbar ridges *I* and *III*; the latter being continuous with the aorticopulmonary septum (A/P septum). The original 270 degree spiral of the bulbar septal anlagen is indicated. A clockwise rotation of the larger circle (ostium V.B.) and a counterclockwise rotation of the smaller one (ostium B.T.) would obviously reduce the amount of spiralling of the ridges. In the normal heart a 45 degree clockwise rotation occurs at the ostium V.B., and a 150 degree counterclockwise rotation takes place at the ostium B.T. The sum of these torsions represents the total reduction of the original 270 degree spiral of the septal anlagen, 195 degrees. When this is subtracted from the original 270 degrees, the remaining 75 degrees are the normal adult torsion of the great vessels about one another. In the interpretation of the present case, an additional 180 degree clockwise torsion is postulated to have occurred at the ostium V.B. due to failure of the fixation of this portion of the bulbus (see text). Following the twisting of the ostium V.B. (45 degrees plus 180 degrees), the remainder of the spiral torsion of the septal anlagen is 45 degrees. This torsion is lost through movement at the ostium B.T. in a counterclockwise direction and explains the nearly parallel course of the great vessels.

The left coronary artery normally develops above the semilunar valve formed from the aortic portion of Ridge I near its junction with Ridge A, while the right coronary artery develops above the semilunar valve related in a similar manner with Ridges III and B. In Fig. 2 these semilunar valves have been labelled A and B, respectively, while their counterparts in the pulmonary trunk are labelled A′ and B′. These may be called the septal valves since they are formed from the ridges which also form the aorticopulmonary septum. Due to the 180 degrees excessive rotation, A and A′ which were once sinistroventral are now dextrodorsal, while B and B′ have reversed their normal position in a similar manner. This explains the nomenclature used for the coronary arteries in Fig. 3.

A similar reversal has occurred in the derivatives of Ridges A and B in the ventricle. In Fig. 2 Ridge A has formed Muscle Ledge A, the crista supraventricularis, normally a derivative of Ridge B. As a result, Ridge B now forms Muscle Ledge B, the trabecula septomarginalis, normally a derivative of Ridge A. Lev and Saphir[51,52] have called Muscle Ledges A and B the parietal and septal muscle bands, respectively.

Explanation of the Aortic Coarctation.—In a recent paper, Bremer[53] has reviewed the classical work of Bonnet.[54] The latter is credited with differentiating between congenital and adult types of aortic coarctation. Among the possible explanations for congenital coarctation he suggested simple arrest in development; complete absence of the interventricular septum causing lessened circulation through the aortic arch; and, last, an involvement of the embryonic aorta in the atrophy of the fifth aortic arch. Bremer reasoned that since adult coarctation is generally considered to be due to the associated vascular degeneration in the ductus arteriosus following birth, the condition of congenital coarctation might logically also be associated with a vascular degeneration at an earlier period of development. Using rat embryos, he found an area of thickening and narrowing of the aorta at the aortic isthmus, and another of the right subclavian artery in an embryologically analagous position. These areas represented the points of attachment of the right and left dorsal aortae between their respective third and fourth aortic arches. He concluded that this produced the aortic isthmus, a narrowed portion of the aorta between the origin of the left subclavian artery and the attachment of the ductus arteriosus. Excessive stagnation of the circulation in this region was postulated to play an important role in the failure of this narrowed portion of the aorta to expand to its normal size. In the present case both of the great vessels arose from a common ventricle and ascended as two parallel channels almost equal in size. There was, therefore, very little pressure differential between these two trunks at the point of the aortic isthmus. Hence, the coarctation is due to the absence of the normal hemodynamic forces operative in this region. Bremer pictures a type of coarctation (Type IV) very similar to the present case in which the left subclavian artery arises from the coarcted segment.

SUMMARY AND CONCLUSIONS

1. A study of a case of atresia (absence) of the mitral orifice has been presented. The unusual combination of anomalies associated with the condition has been described in detail.

2. The literature on forty-eight other cases of congenital mitral atresia or absence has been studied, and of these, forty-two have been included in this analysis.

3. An interpretation of the present case is presented, based on the work of others and the unusual conditions observed in this case.

4. The congenital aortic coarctation present in this case was so unusual that it deserves special consideration.

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CONGENITAL GLYCOGENIC TUMOR OF THE HEART

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SIXTY-FOUR cases of congenital glycogenic tumor of the heart have been reported in the literature up to 1947.[1] Because it involved interesting diagnostic problems that were confirmed by post-mortem examinations, we now present the following case.

CASE REPORT

One day after birth the male infant of a 34-year-old white woman was seized with cyanotic spells which increased in intensity. This was the mother's second pregnancy. Her prenatal course was uneventful, and the child appeared normal at delivery. Her first child was normal with no congenital defect.

Dr. Irving J. Wolman, the consulting pediatrician, gave the following opinion: "Baby has cleft palate and is mildly icteric. Was cyanotic last night. Placed in incubator. Has precordial bulge. Heart rate slow (about 60); somewhat irregular with extrasystoles on inspiration. Sounds loud and forceful. First sound accompanied by faint murmur on occasion. No thrills. Examination otherwise negative. Not cyanotic at this time. Impression: Congenital cleft palate and heart lesion."

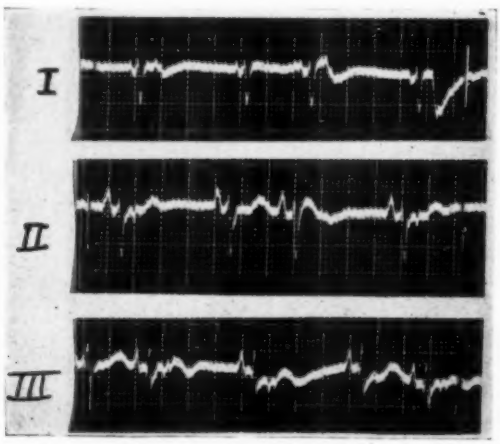

Fig. 1.—Electrocardiogram. The most significant change in this electrocardiogram is the presence of an arrhythmia that appears to be the result of frequent interposition of blocked auricular premature contractions. In so young an infant such an arrhythmia is most infrequent. Its significance cannot be stated with any finality at this time; it may be an expression of myocardial ischemia. The only potential hazard from this type of arrhythmia is the development of an auricular paroxysmal tachycardia.

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Fig. 2.—Gross picture of the nodule at the right atrium.

Fig. 3.—Inside view of the right ventricle and atrium with the tricuspid valves. Note the well-demarcated nodule on the outer surface of the right atrium.

In cardiologic consultation, Dr. Martin H. Wendkos reported: "During the first twenty-four hours of life a definite cardiac arrhythmia was present. An electrocardiographic study indicated that the arrhythmia was the result of frequent auricular premature contractions, some of which were blocked. This phenomenon resulted in an arrhythmia which clinically could not be distinguished from auricular fibrillation since the premature contractions did not occur with any regularity. There were no indications of cardiac failure. Such unusual arrhythmia in a newborn suggested the presence of some toxic factor or inflammation or invasive lesion of the auricles" (Fig. 1).

The cyanotic episodes increased in intensity. The child had fever, showed signs of dehydration, and died five days after birth.

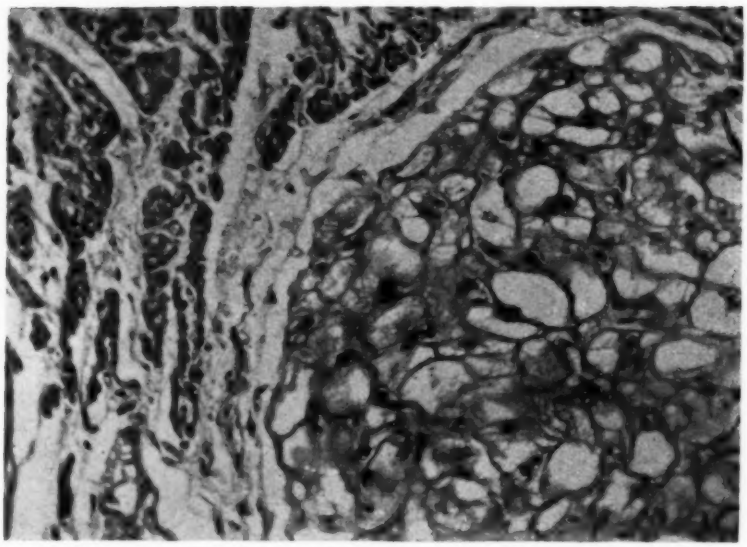

Fig. 4.—Photomicrograph of the nodule of the heart revealing the sharp demarcation of the nodule which is composed of vacuolated spaces with the "spider cells." Hematoxylin-eosin stain (× 200).

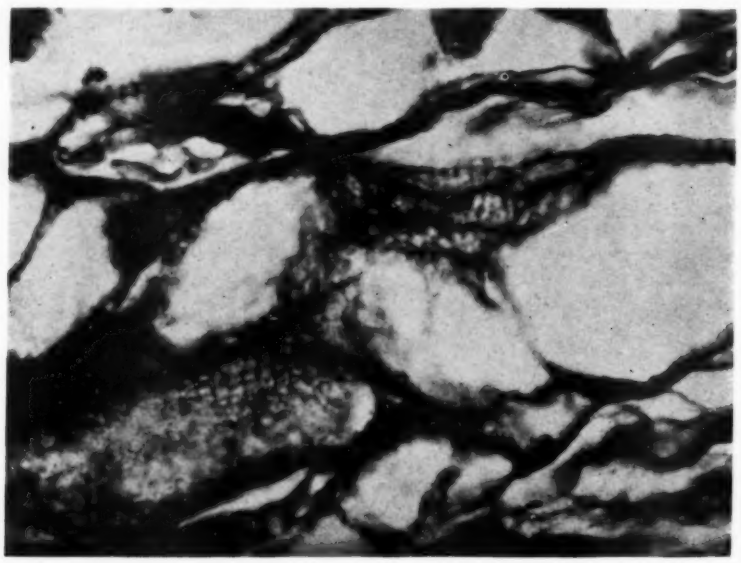

Fig. 5.—Photomicrograph of the cells of the nodule. This power view reveals the presence of transverse striations in the cytoplasm stained with Mallory's phosphotungstic acid indicating the striated muscle cell nature of these cells (× 1,100).

Autopsy Description: The body was that of a well-developed, poorly nourished 5-day-old white infant measuring 56 cm. in length and weighing 3,550 Gm. The visible mucous membranes and skin showed a yellow discoloration. The mandibula was poorly developed. The soft palate was open, revealing a slitlike opening in the middle line extending from the gum to the hard palate. Pericardial surfaces were pale pink-gray and glistening.

The heart appeared to be enlarged, measuring 4 by 4 by 3 cm. Several nodules were present in the myocardium, protruding above the epicardium, the largest of which was in the right atrium

measuring 1 by 0.8 by 0.8 cm. Smaller nodules were present over the left ventricle along the sulcus; these were firm and pale gray-yellow with a uniform yellow-gray cut section (Figs. 2 and 3). One nodule appeared to be breaking into the lumen of the arteria pulmonalis producing irregular gray elevations up to 0.3 cm. in diameter just above the semilunar valves. The chambers were distended but the valves appeared normal.

The lungs were enlarged and dark gray-red. Beneath the pleura there were irregular hemorrhagic areas measuring up to 0.5 cm. On section of the lower lobes, which were firm and indurated, there were irregular, confluent, pink-gray granular areas measuring up to 1 cm. in diameter. The lumen of the bronchi contained hemorrhagic frothy liquid. The liver, kidneys, and spleen showed a moderate degree of venous congestion. The adrenal glands revealed the presence of bloody liquid in their medullary portions. The brain was normal in size with the usual configuration. On routine sections no abnormal foci were noted.

Gross Anatomic Diagnosis: Multiple congenital tumor nodules of the heart; cleft palate; hypoplasia of the mandibula; bilateral hemorrhage of the adrenal medulla; terminal bronchopneumonia.

Histologic Examination of the Nodules of the Heart: The nodules were composed of vacuolated spaces varying in size and shape. There were "spider cells" present (Fig. 4). The special Best carmine stain revealed that most of the vacuolated spaces contained bright red granules indicating the presence of glycogen. The Mallory phosphotungstic acid stain revealed the presence of transverse striations in the cytoplasm (Fig. 5). These "spider cells" were interpreted as striated muscle cells which became vacuolated by the intracellular accumulation of the glycogen in such a manner that the nucleus of the cells remained centrally located, surrounded by a narrow zone of cytoplasm assuming the spider pattern.

DISCUSSION

There are several reviews of the literature on congenital tumor of the heart, the last, by Batchelor and Maun,[2] appearing in 1945. Congenital tumors have been called rhabdomyoma, a misnomer firmly fixed in the literature. Since the true neoplastic nature of the tumors has been questioned, the term congenital nodular glycogenic degeneration of the myocardium is also suggested. This, however, is not accurate because there is no proof that an accumulation of glycogen in the myocardial fibers represents a degenerative phenomenon. The term hamartoma, as used by Albrecht,[2] is more suitable since it indicates the congenital nature and the lack of neoplastic qualities of the nodular lesion. Still, it does not emphasize the most important feature of the entity, the glycogen content. The reviewers suggested the term congenital nodular glycogenic tumor of the heart.

Histologic study of the nodules of the heart confirmed that this condition is a hamartoma or tumorlike malformation consisting of localized misplaced striated muscle tissues with abnormal glycogen deposition; it is not a neoplastic condition.

SUMMARY

A case of congenital glycogenic tumor of the heart has been presented with an electrocardiographic study made on the second day of life. On the basis of our histologic studies, we believe that this is not a neoplasm but represents a hamartoma or tumorlike malformation consisting of misplaced striated muscle tissue with glycogen deposition.

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SUDDEN DEATH DUE TO EMBOLIC OCCLUSION OF BOTH CORONARY OSTIA IN SYPHILITIC CARDIO-VASCULAR DISEASE

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SUDDEN death in syphilitic heart disease has been considered the result of a ruptured aortic aneurysm, coronary ostia stenosis, or sudden disorders of rhythm due to myocarditis or gummas. The following report deals with an apparently rare complication of syphilitic aortitis which is another pathological phenomenon for the cause of sudden death as well as an hitherto unrecognized end result of syphilitic heart disease.

Embolization to the coronary arteries is an unusual circumstance under any situation, and up to 1933 there were only thirteen well-documented cases from a variety of sources.[1] The pathogenesis of such emboli has been from air,[2] tumor,[3] fat,[4] infected thrombotic materials[5a, b] as in subacute bacterial endocarditis, paradoxical emboli,[6] and now from mural thrombi from the aorta. The following two cases of syphilitic aortitis with mural thrombi and subsequent embolization were observed among approximately 75,000 consecutive autopsies performed at the offices of the Chief Medical Examiner of the City of New York.

CASE REPORTS

Case 1.—A 30-year-old Negro woman was found dead in bed. The available history elicited from her husband reported the complaint of occasional dyspnea on exertion. There was no information as to history or treatment of venereal disease.

Autopsy.—

Heart: The heart weighed 450 grams. The epicardium was smooth and shiny, and the myocardium was beefy-red and firm. The left ventricle measured up to 1.8 cm. and the right ventricle up to 0.5 cm. The left ventricle and auricle were considerably dilated, and the papillary muscles and chordae tendineae were thickened and flattened. The endocardium was smooth and shiny. The mitral, tricuspid, and pulmonary valves were grossly normal. The aortic valve measured 68 mm. at the ring and showed considerable thickening of the closing margin of the cusps. There was separation of the commissures between the posterior and left anterior leaflets and between the right and left anterior leaflets. Below the latter commissure there was a small endocardial pocket. The ascending aorta showed wrinkling and fissuring of the intima alternating with raised, small, pearly opalescent areas. Here and there small "punched-out" scars surrounded by radiating lines were noted. Small, yellowish, atheromatous plaques were scattered throughout this portion of the intima. These findings decreased in intensity as the aorta was traced to its bifurcation. On the right posterior lateral wall of the aorta, 2 cm. above the aortic ring, there was a saccular aneurysm measuring 12 mm. in diameter which penetrated into the pulmonary artery

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eroding its wall up to the intima but without evidence of perforation (Fig. 1). The aneurysm contained a large, soft, lamellated blood clot which at its lower border protruded across a thick ridgelike projection about 3 cm. in length into the lumen of the aorta where it was loosely attached to the wall. Small, firmly wedged blood clots occluded both coronary orifices (Fig. 2). After removal of the clotted material, both ostia were seen to be narrowed to about one-half the normal diameter.

Of the remaining organs, the liver and spleen were surrounded by thin fibrous adhesions, and both tubes presented evidence of chronic salpingitis and hydrosalpinx.

Microscopic Examination.—

Aorta: The wall of the aorta was thickened. The intima of the vasa vasorum revealed considerable proliferation, thus producing narrowing of their lumina. Their adventitia was infiltrated with lymphocytes and plasma cells. The adventitia of the aorta showed dense perivascular infiltration with similar cells which extended frequently into the media. Occasional foci of necrosis were noted in the media corresponding to the interruption of the lamellae of elastic fibers as seen in the Van Gieson-Elastica stain. In several areas, small gummas were noted which involved the media as well as the innermost layers of the adventitia (Fig. 3). Occasional fibrous replacement of the elastic tissue and muscle fibers of the media was noted. The intima showed the deposition of large foci of hyaline material surrounded by pale, loose, connective tissue containing a frayed internal elastic membrane.

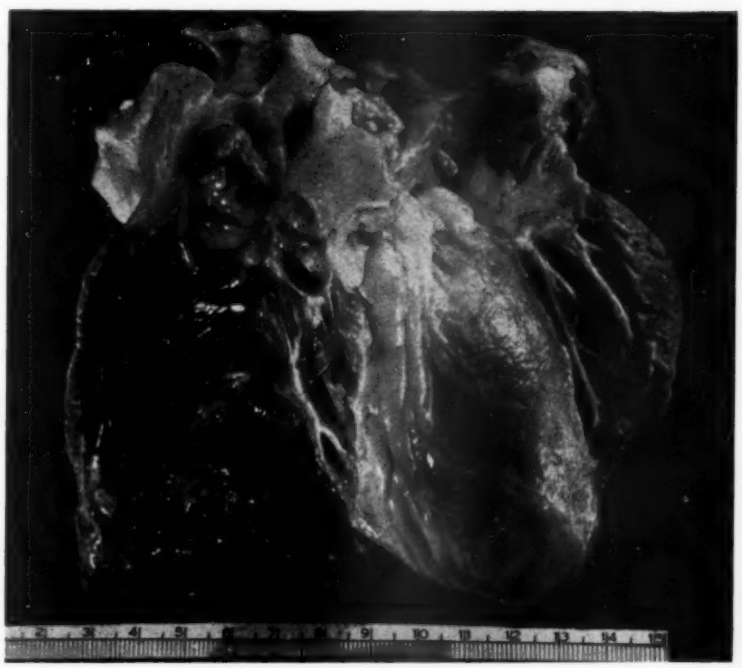

Fig. 1.—Pulmonary artery; a saccular aneurysm of the aorta penetrates into the pulmonary artery.

Sections from the aneurysm showed essentially the same histologic pattern. At one point in the penetration the pulmonary artery consisted only of the intima and innermost layers of the media.

Heart: The epicardium and subepicardial fat showed diffuse, dense infiltration with lymphocytes and plasma cells mainly at the base of the heart. In these areas the infiltrating cells occasionally penetrated the outermost portions of the heart muscle. The muscle fibers of the left

ventricle showed hypertrophy, and the cusps of the aortic valve were thickened at their closing margin, showing an increase of collagenous ground substance with occasional round-cell infiltration.

Coronary arteries: Both coronary arteries showed syphilitic changes in the first 5 mm. There was diffuse infiltration of their adventitia with lymphocytes, plasma cells, and histiocytes. The ostium of the right coronary artery showed occasional miliary gummas of its media which, in one area, extended into the intima. In several places the inflammatory cells of the adventitia of both coronary arteries invaded the media; the elastic fibers with special stain were frayed and split. Occasional small atheromatous foci were seen in the remaining portions of the coronary vessels. Levaditi stain did not show the presence of *Spirochaeta pallida*.

CASE 2.—A 50-year-old white man injured his right ankle, which was then placed in a cast. While waiting for an x-ray, he complained of sudden precordial pain and shortness of breath. While the roentgenogram was being taken, the patient died. There was no positive history of syphilitic infection or treatment.

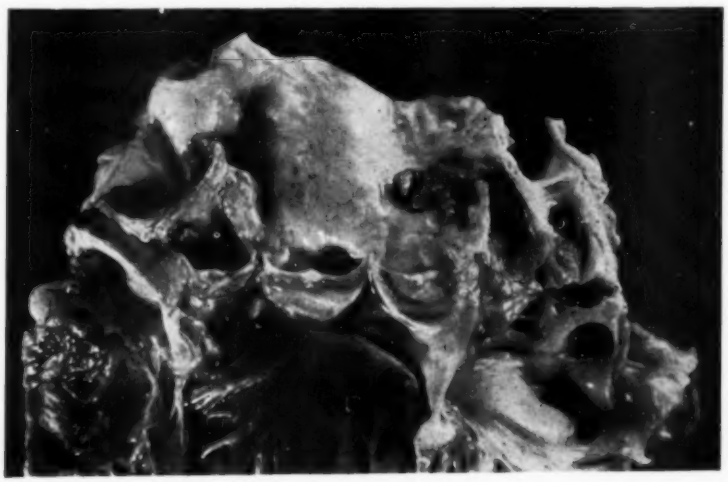

Fig. 2.—Syphilitic aortitis. Both coronary ostia are occluded by emboli. There is the opening of an aneurysm in the right upper corner containing a thrombus.

Autopsy.—

Heart: The heart weighed 370 grams. The epicardium was smooth and shiny. The left ventricle measured up to 1.5 cm. in thickness and the right ventricle up to 0.3 cm. The myocardium was beefy-red and firm. The papillary muscles and chordae tendineae were flattened. The edges of the aortic valve were thickened and rounded, and there was separation of the commissures. The remaining valves were grossly normal. The ascending aorta showed wrinkling, scarring, and fissuring of the intima, interspersed with frequent small pearly-gray areas. There was a large aneurysmal dilatation of the ascending aorta. The intima showed extensive yellowish atherosclerotic plaques which were occasionally replaced by shallow ulcers. One of these ulcers was noted at the sinus of Valsalva, 1.5 cm. distal to the orifice of the left coronary artery. To this ulcer was attached a thrombus measuring 15 by 11 by 6 mm. The tip of this thrombus blocked the orifice of the left coronary artery. The orifice of the right coronary artery was also occluded by an embolus which was firmly wedged into its orifice. After removal of both blood clots the diameter of the ostia was seen to be decreased by about 50 per cent. Both arteries were patent throughout their remaining course. There was moderate atherosclerosis of the abdominal aorta.

The remaining organs were grossly normal. Microscopic studies were not performed, but a Kline test done at the time of autopsy was positive.

DISCUSSION

In 1940, Porter and Vaughan[7] reviewed 3,000 autopsy records of the Medical College of Virginia and found three cases of coronary embolism. All three patients had syphilitic heart disease with aortitis with mural thrombi of the aorta. In all instances the left coronary artery was occluded. They collected thirty cases of well-documented coronary emboli from the literature, including their own, of which twelve were due to endocarditis, seven to aortic thrombi, three to syphilitic aortitis, two to ventricular thrombi, two to thrombus of the coronary artery, and four to paradoxical emboli. Of the thirty, twenty-three died suddenly. The majority of deaths were due to occlusion of the left coronary artery,

Fig. 3.—Syphilitic aortitis. The adventitia and areas of the media are diffusely infiltrated by lymphocytes and plasma cells. In the center a small gumma is noted. Hematoxylin and eosin, × 150.

but in four instances both coronaries were involved. The source of the bilateral occlusions were tumor tissue, pedunculated thrombi on the wall of the aorta, and two cases of endocarditis. In their own cases they attributed atherosclerosis of the aorta as the cause of the mural thrombi. In 1940, Hamman[8] reviewed thoroughly the problem of coronary embolism and made particular reference to the frequent incidence of coronary embolism secondary to mural aortic thrombi. He was able to find six cases of thirty-eight well-documented coronary emboli due to the formation of thrombi on the aortic wall. This was causally second in incidence to subacute endocarditis. He added one case due to syphilitic aortitis,

where there was bilateral embolization. Pratt-Thomas[9] described an additional case in a 32-year-old Negro, where a large thrombus secondary to syphilitic aortitis occluded the right coronary artery. At this point there were five cases described in the literature of coronary embolization secondary to mural thrombi of syphilitic aortitis. All of these patients and those to be described later were in relatively young people.

Greenstein[10] reported five additional cases of syphilitic aortitis complicated by mural thrombi and embolization, the right coronary artery being involved twice and the left coronary artery three times.

The anatomic findings in the cases reported in this paper suggest the following sequence of events: The first case developed a saccular aneurysm of the aorta, containing a large thrombus, penetrated into the pulmonary artery. Blood clots detached from the thrombus and under a specific hemodynamic situation became firmly wedged into both narrowed orifices. In the second case, there was considerable atheromatosis with ulceration secondary to syphilis with the formation of a mural thrombus. The tip of the thrombus occluded the left coronary ostium and an embolus occluded the right coronary ostium. This is similar to the situation in Hamman's case. In the first case the mesaortitis was specific for syphilis, regardless of the noncontributory Levaditi stain. In the second, in addition to the characteristic gross changes, the Kline test was positive.

With the present report there are twelve cases of coronary embolization due to mural thrombi arising from syphilitic aortitis or aneurysm secondary to aortitis. The mechanism is certainly not clear. The suggestion that aortic regurgitation with its resultant back flow of blood during the diastolic period of filling of the coronaries is not completely tenable. Certainly the incidence of mural aortic thrombi due to arteriosclerosis or syphilitic aortitis is more frequent than coronary embolization under such circumstances. Apparently very specific hemodynamics must be present for the catastrophe of embolization, the nature of which is unknown.

Despite the absence of such lesions in large groups of syphilitic hearts examined,[11] one must add to the usual causes of death in syphilitic heart disease the catastrophe of coronary embolization.

SUMMARY

1. Two cases of syphilitic aortitis with mural thrombi complicated by bilateral coronary embolization are presented.

2. The literature of this subject is reviewed, and mention is made of the necessity of including this pathologic entity as an infrequent cause of death.

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Editorial

THE study of the heart and circulation in health and disease undoubtedly began before recorded history. There are, however, delineated, as we look backward, certain eras in this history which stand out in bold relief. It is not our purpose at present to review all of these in equal detail. For the sake of convenience, these eras may be identified by the name of an outstanding individual of his time and also by what principally distinguished the era.

The first era may be called that of anatomic discovery, closely associated with the name of Vesalius, but in this era there were also equally prominent men, such as daVinci, Paré, Fallopius, Eustachius, Fabricius, Servetus, and many others. That they understood the anatomic features of the heart and circulation there can be no doubt, but they did not appreciate the functional importance of many of their discoveries.

It remained for the second era, or what may be called the introduction of the experimental method as led by Harvey, to interpret these anatomic discoveries. There is no doubt that Harvey learned about the valves of the veins from Fabricius, but the latter could not conceive of their proper function. Harvey's enunciation of the circulation on a functional or what might be called today a physiologic basis is commonly considered to be the beginning of modern cardiology. This era, however, was not without other men who made important contributions. Among these may be mentioned Lower, Stephen Hales, and others.

In spite of these advances in anatomy and physiology, the means whereby the circulation could be interpreted at the bedside were scant indeed. It is true that certain information was obtainable through the examination of the pulse, and Morgagni had described heart block with its epileptiform sequelae. It was not until Laënnec published his treatise on mediate auscultation that the third era may be said to have begun; it may be called by his name, or that of physical examination and diagnosis. It is true that Auenbrugger had published his observations on percussion of the chest some fifty-eight years before, but they remained practically unnoticed. It required auscultation to make progress in this era really effective. Other names that come to mind are: Adams, Corrigan, Stokes, Duroziez, Flint, Traube, Broadbent, and a host of others. Parallel with this great upsurge of clinical observation there was the growth of the school of pathologic anatomy under the great Virchow. This led to the domination of fixed anatomic lesions over the functional requirements of the circulation. It was a period of therapeutic nihilism, and even the discovery by Withering of the value of digitalis had fallen into disrepute and neglect.

From this background of functional stasis, men's minds began to be disturbed and to revolt against the futility of much of medical practice. True, the discoveries of antisepsis and anesthesia had given to surgery the tools for its

phenomenal advance. Bacteriology also gave a new meaning to public health and to the causation of the fevers. But the fourth era for the exploration of diseases of the circulation was at hand. This may be called the era of clinical physiology. In 1883, James Mackenzie, a general practitioner in Burnley who had graduated five years previously from the University of Edinburgh, began his long and tedious work with a Dudgeon's sphygmograph on the pulse and cardiac rhythm. It was not until 1890 that he reached the point of being able to unravel the different types of cardiac irregularities. The first one that he discovered was ventricular extrasystoles; the next he called childhood irregularity, what we know today as sinus arrhythmia; and finally, he identified what he first designated as the irregularity of danger and later called nodal rhythm, which in our time is known as auricular fibrillation. It was about this time that, with the aid of Mr. Shaw, a clock-maker in Burnley, he evolved what was later to be known as the writing polygraph. Now Mackenzie had in his hands an instrument which suited his purpose. He could take multiple simultaneous tracings of the radial, apical, or jugular pulsations, or any combination thereof. Furthermore, instead of the short and messy strips of the sphygmograph, he could take tracings for minutes or hours. It was not, however, until 1902 that he published his first work entitled "The Study of the Pulse," to be followed, in 1908, by his larger volume, "Diseases of the Heart." It is interesting to note that Traube, in 1872, had published a description of a case of pulsus bigeminus. Also, Byrom Bramwell, in his "Studies in Clinical Medicine" (1889-90, Division 1, p. 115), published a sphygmographic tracing in the case of a young man with a pulse rate of 51 in which he recognized a small pulse between every two normal pulsations. This occurred with *absolute and undeviating regularity*. Some days later he reported that the pulse numbered 102 to 104 per minute, exactly double its previous rate. Continuing his clinic, he states, on page 118: "The cardiograph and sphygmograph do, I believe, afford useful information in some cases of cardiac and arterial disease. Their value is sometimes no doubt exaggerated . . . I have no sympathy with those persons who call themselves practical men, and who pooh-pooh the importance of these and many other instrumental means of clinical research. Every additional means of research has its value; only, in using these means of research, one must be careful to attach the correct value, and not an exaggerated value, to the results which we obtain by their use." Bramwell was apparently voicing a criticism of the chauvinism of the times, as Mackenzie's work was not really accepted in Great Britain, except by a few, until after he came from Burnley to London in 1907, although it had been well known and accepted and had stimulated others in Europe and America from 1902.

There were, however, at this time, other scientists approaching myocardial function from a different direction. In 1887, Augustus R. Waller published in the *Journal of Physiology* an article entitled "A Demonstration on Man of Electromotive Changes Accompanying the Heart-Beat." It attracted but little attention, nor was this surprising as it was such an heretical concept. The instrument did not come into common clinical use because much difficulty was encountered with it. In 1903, William Einthoven, using his string galvanometer, published his paper "The Galvanometric Registration of the Human Electrocardiogram,"

likewise a review of the use of the capillary electrometer in physiology. He followed this by many articles dealing with electrocardiography. On March 19, 1912, he delivered a lecture before the Chelsea Clinical Society in London, which was published in the *Lancet* the same year. Then came, in 1913, a further article, in collaboration with Fahr and deWaart, in which he outlined his well-known triangle for calculating the electrical axis of the heart (Pflüger's *Arch. f.d.ges. Physiol.* **150:** 275-315, 1913). It would seem at first glance that these two publications are identical.

There are very few cardiologists in North America who have had an opportunity of reading the 1913 article in the original. In fact, there are not many who know of the publication in the *Lancet.* To meet this situation, the Editorial Board have, in this issue, published a translation of the 1913 article. It will be seen that this translation has been most ably done by Professor Hebbel E. Hoff and Dr. Paul Sekelj, and we wish to take this opportunity to express to them our sincere thanks.

It was mentioned above that the publications of 1912 and 1913 might appear to be identical or at least substantially similar, but the translators point out that this is not the case. In order that one may be compared with the other, they have compiled a concordance of the paragraphs and of the illustrations in both. These are published as addenda to the translation to assist those interested in comparing the two publications. As the translators state in a footnote to the concordance of the paragraphs: "The concordance is at times exact, with no changes other than different paragraphing. At other times, however, a single sentence in the *Lancet* article is expanded into several paragraphs in the Pflüger's article, and new illustrative material is added. The impression is clear that the *Lancet* article was written first, and was reorganized, edited, and enlarged for publication in Pflüger's *Archiv.*"

It is our sincere hope that this translation will be of assistance to students of electrocardiography.

J.C.M.

Notice

The first meeting of the recently founded International Society of Angiology will be held in Paris, Sept. 4 and 5, 1950, at the Collège de France, Place Berthelot. The officers of this society are: Dr. René Leriche (Paris), President; Drs. Emile Holman (San Francisco), Alexander M. Boyd, (Manchester), and Fernando Martorell (Barcelona), Vice-Presidents; and Dr. Henry Haimovici (New York), Secretary. All correspondence regarding the Society and its first meeting should be addressed to Dr. Henry Haimovici, Secretary, 1148 Fifth Avenue, New York 28, N. Y.